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ANATOMIC AND PHYSIOLOGIC STUDIES OF THE EIGHTH NERVE

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Although many investigations have been conducted on the eighth nerve, there still remain numerous questions both as to the anatomy and physiology that have not been solved or about which there is some difference of opinion. In the last few years the interesting clinical observations of Bárány and Neumann [1] in Vienna, and Jones and Mills [2]

1. Bárány, R.: Ueber dem vom Ohre auslösbaren Nystagmus Vortrag a. d. 77, Vers. deutsch, Naturforsch. u. Aerzte Ztschr. f. Ohrenheilk. **51:** No. 1. Vestibular Erkrankungen und Neurose. Vortrag. geh. in Neurologische Gesellschaft zu Wien, Jan. 9, 1906, Wien. klin. Wchnschr. 1906, p. 489. Untersuchungen über den vom Vestibular Apparat des Ohres reflectorisch ausgelösten rhythmischen Nystagmus und seine begleitende Erscheinungen. Monatschr. f. Ohrenheilk. **11:**193-297, 1906. See also Ebenda, 1907. Beitrag zur Lehre von den Funktionen der Bogengänge, Ztschr. f. Psychol. u. Physiol. d. Sinnes Organen **41:** Pt. 2, 37-44, 1906. Ueber die vom Ohrlabyrinth ausgelöste Gegenrollung der Augen bei Normalhörenden, Ohrenkranker und Taubstummen, Arch. f. Ohrenheilk. **68:**1-30, 1906.

2. Mills and Jones: Neuraxial Differences of Fibres from Horizontal and Vertical Canals Determined by Bárány Tests, J. Nerv. & Ment. Dis. **44:**245, 1916.

and others in this country, have made the subject a particularly enticing field for further studies. Both the European and American observers have deduced anatomic and physiologic facts from their clinical observations. In some cases they have assumed the existence of anatomic pathways in order to explain adequately their clinical findings. The Bárány

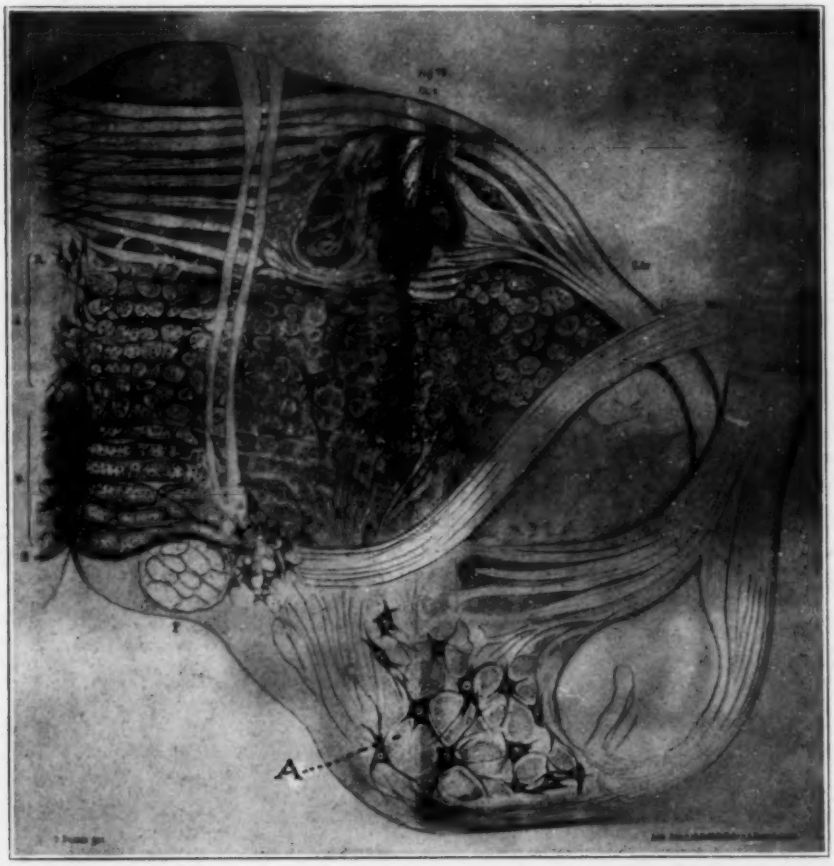

Fig. 1.—Illustration from Schultze's book on Deiters' work. This is Deiters' original drawing and shows that what is now called von Bechterew's nucleus is the nucleus Deiters originally described, and which Schultze named Deiters' nucleus.

enthusiasts have gone so far that many of them believe to be facts what a few years ago were only theories. The purpose of the present investigation has been to study by modern and somewhat different methods the eighth nerve mechanism with special reference to the vestibular portion.

REVIEW OF LITERATURE

One part of the physiologic phase of the subject has been completely reviewed up to 1894 by von Stein [3] in his monumental book of almost 700 pages. Most of the book, however, is devoted to the vast amount of work that has been done on the cochlea and semicircular canals. The book contains little mention of the work on the central connections of the eighth nerve.

As our studies have been along anatomic and physiologic lines, we shall not review the work of Bárány, Neumann, Jones [4] and those

Fig. 2.—Experiment 135. The eighth nerve, *N VIII*, is completely cut across. Degenerated vestibular fibers, *VE*, are seen running to Deiters' nucleus, *D*. The arcuate fibers, *ARC*, are well seen. Note the slight injury to the adjacent cerebellum.

others who have dealt with the eighth nerve clinically. We shall briefly summarize the important points that have been described by previous observers which bear on this investigation. These articles will be taken up in chronologic order.

3. Von Stein, Stanislav: Die Lehren v. d. Functionen der einzelnen Theilen des Ohrlabyrinthes zusammegestellt von S. v. Stein, deutsch von Krzywicky. (Verlag G. Fischer, Jena).

4. Jones, I. H. and Fisher, L.: Equilibrium and Vertigo, Philadelphia, Lippincott Co., 1918.

Von Monakow [5] (1882) made this statement: "An isolated extirpation of the eighth nerve for the purpose of studying the secondary atrophy is hardly feasible because of the impossibility of avoiding injury to neighboring parts."

Von Bechterew [6] (1885 and later) described the following symptoms when the posterior cerebellar peduncle was cut: "rolling over and over, circus movements in the long axis of the body." When the eighth nerve was cut on one side, the animal rolled to the side of the operation. When the region of the aqueduct of Sylvius or the anterolateral portion of the fourth ventricle was cut, the animal rolled to the

Fig. 3.—Experiment 92. Vestibular fibers, *VE*, going to Deiters' nucleus, *D*.

opposite side. Nystagmus was present in all experiments. The animals lived only one or two days after operation. The specimens were studied by the Weigert method. There are no drawings or photographs to show the lesions. He concluded that the tracts from the olive and vestibular nerve to the cerebellum passed up the posterior peduncle,

5. Von Monakow: Experimentel Beitrag zur Kenntnis d. corp. Restiforme, Arch. f. Psychiat. **14**: No. 1.

6. Von Bechterew: Ueber die Verbindungen der oberen Oliven und ihre wahrscheinliche physiologische Bedeutung, Neurol. Centralbl., 1885, No. 21. Zur Anatomie der Schenkel des Kleinhirns, ibid., p. 121. Zur Frage über d. Ursprung des Hörnerven über d physiolog. Bedeutung des N. Vestib., ibid., 1887, p. 193

also that the olivary tract fibers decussate in the cerebellum. He was one of the first to work on the connections between the semicircular canals and the cerebellum.

Onufrowitsch,[7] working with Forel, studied the brains of two rabbits on whom at birth one otic bone had been destroyed. They studied the brains by noting the secondary atrophy and found that the vestibular fibers ended in the tuber acousticum.

Baginsky[8] (1885 and 1886) expressed the view that the symptoms von Bechterew described were due not to injury to the eighth nerve, but to lesions of the neighboring parts of the brain. In his own experi-

Fig. 4.—Vestibular fibers running to Deiters' nucleus. This illustration shows the amount of degenerated fibers, *PF*, resulting from retraction of the paraflocculus.

ments he obtained the same symptoms and demonstrated lesions of the brain. In a later article he pointed out that only the posterior part of the eighth nerve is connected with the cochlea. Fibers from this portion pass to the tubercle and in part circle the corpus restiforme dorsalward. Many fibers also pass ventrally as arcuate fibers to the opposite side. He believed that only the anterior of the three acoustic

7. Onufrowitsch: Experimenteller Beitrag zur Kenntnis des Ursprungs des Nervus Acusticus, Arch. f. Psychiat. **16**:3.

8. Baginsky: Zur physiologie du Bogengänge, Arch. f. Physiol, 1885, p. 252. Ueber d. Ursprung u. d. centralen Verlauf d. Nervus akust. d. Kaninchens, Sitzb. d. k. preussischen Akad. d. Wissenschaft **25**: (Feb.) 1886.

nuclei belonged to the posterior root. Fibers also go to the posterior corpus quadregeminum and internal geniculate body. There is no connection between the root and the cerebellum.

Bumm [9] (1889), working on rabbits at birth, believed that the posterior root of the eighth nerve had no connection with the cerebellum. The anterior acoustic root arises from the medulla and part of the cerebellum. Its origin is at the ventral border of Deiters' nucleus and in the vermis.

Biedl [10] (1895) used the Marchi method. His animals showed circus movements, rolling over and over and forced extension movements.

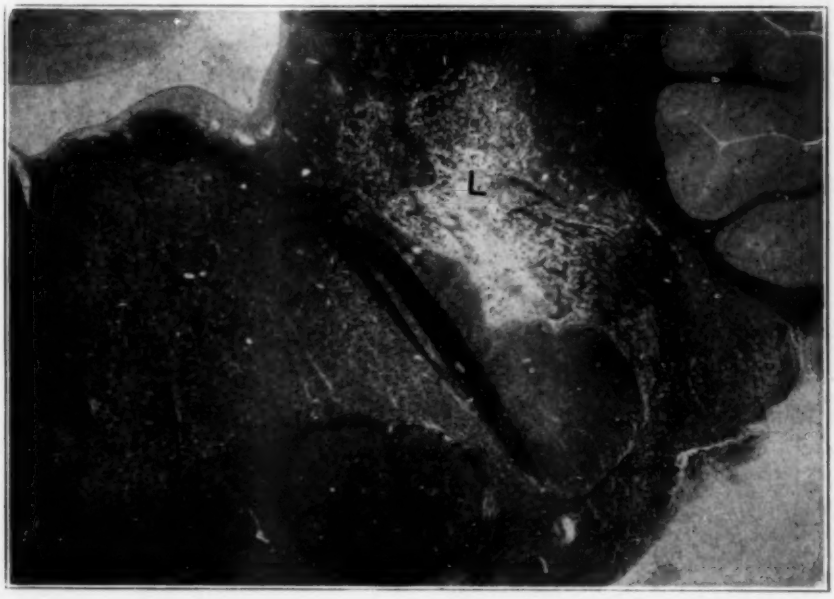

Fig. 5.—Experiment A5. Lesion, *L*, of Deiters' nucleus and Bechterew's nucleus.

Fibers pass to both posterior longitudinal fibers; others pass by the way of the arcuate fibers to the other side to form the lateral lemniscus; a third group pass to the olive of the opposite side and travel centrifugally.

Ferrier and Turner [11] (1894, 1895) sectioned the eighth nerve in monkeys. They observed rolling movements, an attitude of the head,

9. Bumm: Exp. Beitr. zur Kenntniss des Hörnervenursprungs beim Kaninchen, Allgem. Ztschr. f. Psychiat. **45**:568-572, 1889.

10. Biedl: Absteigende Kleinhirn-bahnen, Neurol. Centralbl., 1895, p. 434.

11. Ferrier and Turner: On Cerebro-Cortical Afferent and Efferent Tracts, Philadelphia Trans., B **190**:1-44, 1898.

extension of the limbs on the opposite side, but no nystagmus. The degenerations, as seen by the Marchi method, showed fibers (1) to Deiters' nucleus, (2) to the outer angle of the fourth ventricle, (3) arcuate fibers to the tegmentum of both sides, (4) to the superior cerebellar peduncle, and (5) to the sixth nucleus.

Oseretzkowsky [12] (1895) made the same observations as the preceding observers, but he emphasized the fact that the arcuate fibers send many fibers to the superior olive of each side and that the decussating fibers from the cochlear nerve are more numerous than those on the same side.

Fig. 6.—Experiment A5. Lesion, *L*, of Bechterew's nucleus.

Thomas [13] (1898) seems to have been the first to cut the eighth nerve intracranially on a dog. He made one experiment. He seems to have studied only the degenerations of the cochlear fibers.

Biehl [14] (1900) experimented on horses and sheep. The horses all died in from one to seven days. Some showed the so-called classi-

12. Oseretzkowsky: Zur Frage vom centralen Verlaufe des Gehörnervs, Arch. f. mikr. Anat. **45**:450, 1895.

13. Thomas: Les terminaisons centrales de la racine labyrinthique, Compt. rend. Soc. de biol., 1898, p. 183.

14. Biehl, K.: Ueber die interkranielle Durchtrenung des N. vestibularis und deren Folgen, Sitzungsberichte der Wiener akad. math.-naturklasse **109**: Pt. 3. (July) 1900.

cal symptoms described by Bechterew. One of his sheep showed none of these symptoms. There was no histologic examination of these specimens.

Fraser [15] (1901) made lesions of Deiters' nucleus and produced vestibular disturbances. He used the Marchi method and showed ascending and descending degenerations on the same and opposite sides. He found fibers going to the posterior longitudinal bundle, which he traced to the third and fourth nuclei and also followed fibers to the anterior corpus quadrigeminum.

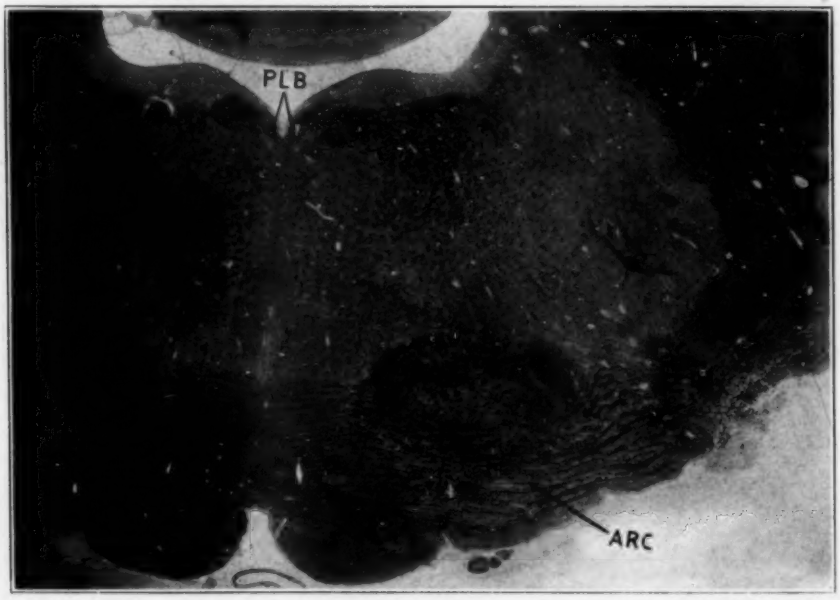

Fig. 7.—Experiment A5. Fibers going to posterior longitudinal bundles, *PLB*, of both sides; arcuate fibers, *ARC*, to superior olive.

Weigner [16] (1903), working on *Spermophilus cititlus*, destroyed the cochlea in some cases with hydrochloric acid, in others with a blunt instrument. He used Marchi, Van Gieson and Weigert stains. His conclusions were:

1. Vestibular fibers pass through Deiters' nucleus.
2. The posterior acoustic nucleus is formed of small cells.
3. Fibers of the nucleus cochlearis pass to the nucleus ventralis and tuberculum acousticum and end there.

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15. Fraser, E. H.: An Experimental Research into the Relations of the Posterior Longitudinal Bundle and Deiters' Nucleus, J. Physiol. **27**:372, 1901.

16. Weigner: Exper. Beitrag. zur Frage v. zentralen Verlaufe d. Nervus Cochlearis bei Spermophilus cititlus, Arch. f. mikr. Anat. **62**:1903.

4. From this gray mass fibers spring which pass dorsally and ventrally.

Van Gehuchten [17] (1903, 1904, 1905, 1906) observed for the most part what our work has confirmed. He asserts, however, that no fibers of the cochlear branch pass to the posterior corpus quadrigeminum; they stop before they get there. He is in accord with Fraser that the fibers in the posterior longitudinal bundle pass to the third and fourth nuclei. He believes that many of the degenerations observed by Tricomi-Allegra [18] (1905) are due to traumatism of other regions during operations.

Fig. 8.—Experiment A5; same section as Figure 7. Fibers running into cerebellum; also fibers to posterior longitudinal bundle, *PLB*. Fibers, *FPF*, from injured paraflocculus can be seen going to lateral lobe and fibers, *T*, from vestibular nerve are seen going to nucleus tecti.

Matte [19] (1907) asserted that he found fibers which pass directly from the membranous labyrinth to the cerebellum.

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17. Van Gehuchten: Recherches sur la voie acoustique centrale, Le nevraxe **4**: 1903. Connexions centrales du Noyau de Deiters et des masses grises voisines, ibid. **6**:21, 1904. La loi de Waller, ibid., 1905, p. 203. Le nerf cochleaire, ibid. **8**:127, 1906. Van Gehuchten and Molhaut: Les lois de la degenerescence wallérienne directe, ibid. **11**:73, 1910.

18. Tricomi-Allegra: Studio sperimentale sulla via acustica fondamentale, Le nevraxe **7**:1905.

19. Matte, F.: Ueber die Frage nach dem Auftreten von sekund. aufsteig. Degen. in. Stamme d. N. Acusticus nach Exstirpation einzelne Teile oder d. ganzen. Häutigen Ohrlab. Zentralbl. f. Physiol., 1907, p. 827.

Wilson and Pike [20] (1912-1913) believe that though the slow component of nystagmus is not affected by removal of the cerebellum or cerebrum, the quick component is affected by the removal of the cerebrum even though the oculovestibular tract is unimpaired. Whether the so-called oculovestibular tract was really unimpaired does not appear from their paper for they report no microscopic examination of their

Fig. 9.—Experiment A5. Fibers, *PCQ*, running from lateral fillet into the posterior corpus quadrigeminum, *CQ*, of opposite side. These fibers come from the lesion in Deiters' nucleus, Figure 6. Nucleus of fourth nerve, *N IV;* no fibers from posterior longitudinal bundle running to it.

specimens, and with the very gross intracranial operations they performed as evidenced by the photographs of their specimens, it hardly seems possible that the oculovestibular tract could have escaped.

20. Wilson and Pike: The Effects of Stimulation and Extirpation of the Labyrinth of the Ear and Their Relation to the Motor System, Philadelphia Trans. Section B **203**:297, 1912.

Thus in their experiment, described on page 145 of their article and shown in Figure 18, it is impossible to assume that at least parts of the superficial fibers were not involved, yet the authors say "There could have been no irritation of the pons as this would tend to cause contraction of the pupil." This hardly seems a justifiable conclusion, for we have injured the pons a number of times without any effect on the pupil.

On the intracranial connections of the two portions of the eighth nerve there are also a large number of important papers. Of these, those by Van Gehuchten [17] and his pupils, Fraser,[15] von Bechterew,[21]

Fig. 10.—Experiment A5. The posterior longitudinal bundle, *PLB*, contains no degenerated fibers. This section is taken somewhat caudalward of fourth nucleus.

Biehl,[14] Probst,[22] Ferrier and Turner,[23] Thomas,[13] Russel, and Sabine, are of greatest interest to us in this particular investigation.

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21. Von Bechterew: Ueber die Verbindung der sogenannten peripheren Gleichgewichtsorgane mit dem Kleinhirn, Pflüger's Arch. f. Physiol. **34**:352, 1884. Ergebnisse der Durchschneid des Acusticus, ibid. **30**:312. Footnote 6.

22. Probst: Ueber vom Vierhügel, von der Brücke und vom Kleinhirn absteigende Bahnen, Deutsch. Ztschr. f. Nervenheilk. **15**:192-219, 1899. Experimentelle Untersuchungen über die Anatomie u. Physiologie der Leitungsbahnen des Gehirnstammes, Arch. f. Anat. u. Physiol. Anat. Abth., 1902.

23. Ferrier and Turner: Experimental Lesions of the Cerebellum and Peduncles in Monkeys, Philadelphia Trans. Section B **185**, II. 1894; also footnote 10.

METHODS USED IN THIS INVESTIGATION

We have performed in all 109 experiments on dogs. Many of these, however, had to be discarded for a variety of reasons. A few were infected, others died from exhaustion due to thrashing about after operation as a result of their violent ataxia and circus movements. In a number of cases the staining was inadequate. This has reduced the cases on which these studies are based to sixty-nine. Some animals operated on with the idea of producing a certain lesion were subsequently transferred to another group after anatomic studies had been completed.

Of these sixty-nine, thirty experiments were made on the semicircular canals. The method described by Wilson and Pike [20] for exposure of the canals was used, but one of us (B. Y. A.) improved on

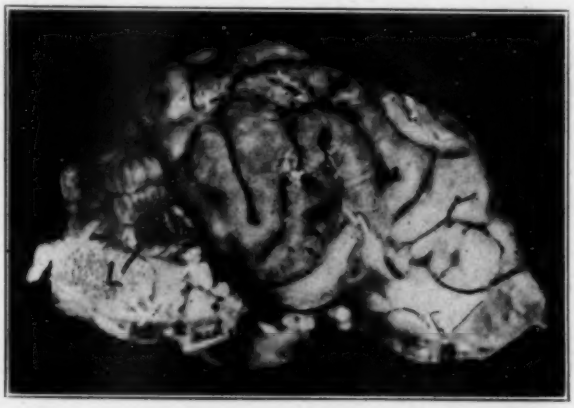

Fig. 11.—Type of lesion, *L*, produced by bone-wax tumors.

it so that he was able to destroy one canal and its ampulla. This was done by opening the canal and introducing a fine wire and destroying the canal and its ampulla. In a few cases all three canals were destroyed by opening one canal and injecting 95 per cent. alcohol stained with eosin. The location of the eosin in the dissection showed that all canals had been destroyed. There were twenty-six cases of pure lesions of the semicircular canals.

In nine cases one canal was destroyed six times, the lateral also called the horizontal, and three times the posterior vertical canal. In fifteen cases both of these canals were destroyed but numbers 48, 55 and 62 showed some degeneration due to opening the intracranial cavity.

In four cases all three canals were destroyed, but one of these showed an injury to the eighth nerve.

In nine cases, while trying to destroy all three canals, hemorrhage occurred from the bone, which was controlled by the use of bone wax.

An opening had been made just medial to the otic bone, and the bone wax projected through the dura and impinged on the cerebellum. These we have called cases of bone wax tumors (Fig. 11). Unfortunately, none of these brains was stained by Marchi, as they were thought not to bear on the problem, but on tabulation of our results it became evident that we had discarded a most valuable set of experiments. Consequently we deliberately made two such bone wax tumors, A 32 and A 33, to study the resulting degenerations.

In twenty-five cases the eighth nerve was cut. In fourteen cases, though an attempt was made to cut only the eighth nerve, more injury occurred, as was revealed by the subsequent histologic studies. In some of these cases Deiters' nucleus was injured, and in some there was a considerable lesion in the cerebellar nuclei; these injuries we believe account for the symptoms the animals presented. We feel justified in this conclusion as our pure eighth nerve lesions did not show these symptoms; Table 1, No. 48; Table 2, Nos. 89, 90, 92, 93, 94, 101, 108, 116, 130, 138, 135. Checking up the gross pathology by careful histologic studies we feel is essential in a research of this sort, and the results of previous observers are confusing and misleading because such studies were not made, at least not recorded in their papers.

In three cases we attempted to destroy Deiters' nucleus with a fine galvanocautery, but none of these lesions show pure Deiters' lesions.

In addition to the sixty-nine experiments on dogs, we have had for study one case in a man on whom we cut the eighth nerve for an intractable tinnitus and numerous clinical observations on the Bárány tests on intracranial cases.

Various methods have heretofore been used to cut the eighth nerve intracranially. The favorite method has been to go through the mastoid bone, pass a hook or knife in blindly and sweep it around. Biehl [13] and Thomas [12] are the only ones who have employed an intracranial approach by retracting the lateral lobe of the cerebellum and Van Gehuchten says he doubts if it can be done.

As one of us had repeatedly exposed the eighth nerve by this route in human beings and was opposed to operative procedures carried out in the dark, this method was used. The operative difficulties, however, are far greater than in human beings on account of the peculiar distribution of the blood vessels. This accounts for our high mortality in this group of animals. This operative approach necessitates retracting the cerebellum slightly, and even the gentlest retraction causes slight degenerative changes, as shown by the Marchi method. All operations were carried out under ether anesthesia.

TABLE 1.—Results of Operation

No. of Exp.	Operation	Nystagmus	Deviation of Eye	Swinging of Head	Attitude	Falling	Rolling	Ataxia	Circus Movements	No. of Days Lived	Stain	Microscopic Findings and Remarks
47	Exposure of otic bone; no canal injured	0	0	0	0	0	0	0	0	18	M	No degeneration
57	Exposure of otic bone; no canal injured	0	0	0	0	0	0	0	0	18	M	Not sectioned
	One Canal											
50	Right posterior vertical	2 →	0	0	0	0	0	0	0	18	M	No degeneration
56	Left horizontal	2 ←	0	0	0	0	0	0	0	18	M	No degeneration
61	Left horizontal	0	0	0	0	0	0	0	0	18	M	No degeneration (sections imperfectly stained)
124	Left horizontal	2 ↷	0	0	0	0	0	0	0	18	M	No degeneration (sections imperfectly stained)
126	Left posterior vertical	0	0	0	0	0	0	0	0	36	N	No chromatolytic changes
127	Left posterior vertical	2 ↑	4	0	0	0	0	0	0	35	N	No chromatolytic changes
132	Left horizontal	2 ←	4	0	0	0	0	0	0	18	M	No degeneration
A31	Left horizontal	2 ↷	4	0	0	0	0	0	0	18	M	Not sectioned
67	Right horizontal	2 →	21	0	0	0	0	0	0	21	M	No degeneration
	Two Canals *											
48	Right posterior vertical and right horizontal	2 ↷	0	0	0	0	0	0	0	18	M	Degeneration of a few fibers of eighth nerve. Skull opened in operation
51	Right posterior vertical and right horizontal	0	0	0	0	0	0	0	0	18	M	Specimen poorly stained
52	Right posterior vertical and right horizontal	1 ↻	0	0	0	0	0	0	0	18	M	No degeneration
55	Left posterior vertical and left horizontal	0	0	0	0	0	0	0	0	18	M	Flocculus destroyed. Fibers to cerebellar lateral nuclei
58	Left posterior vertical and left horizontal	0	0	0	0	0	0	0	0	18	M	Poorly stained
62	Left posterior vertical and left horizontal	2 ↷	18	0	2	0	0	5	0	18	M	Skull opened. Evidence of some intracranial injury. A few degenerated fibers in the pons (slight peduncular lesion)
63	Left posterior vertical and left horizontal	0	0	0	12	0	0	0	0	18	M	Specimen not properly penetrated
65	Left posterior vertical and left horizontal	0	0	0	0	0	0	0	0	18	M	Specimen poorly stained
72	Right posterior vertical and right horizontal	1 ↻	10	0	0	0	0	0	0	18	M	Poorly stained because of formaldehyd
73	Right posterior vertical and right horizontal	0	12	0	0	0	0	0	0	22	M	Not examined, as poorly preserved
76	Left posterior vertical and left horizontal	2 ↷	7	0	0	0	0	0	0	7	M and N	No degeneration and no chromatolytic changes
121	Left posterior vertical and left horizontal	2 ↷	28	0	0	0	0	0	0	28	M	No degeneration
133	Left posterior vertical and left horizontal	1 ↷	0	0	0	0	0	0	0	18	M	Specimen not examined
138	Left posterior vertical and left horizontal	2 ↷	0	0	0	0	0	0	0	18	N	No chromatolytic changes
131	Left posterior vertical and left horizontal	0	0	0	0	0	0	0	0	21	M	No degeneration
	Three Canals											
80	Left side	2 ←	Present	0	0	1	0	0	0	56	N	No chromatolytic changes
125	Left side	0	0	0	0	0	0	0	0	49	N	No chromatolytic changes
129	Left side	2 (?)	0	0	0	0	0	0	0	18	M	Slight injury of eighth nerve and degenerated fibers as seen in all cases of eighth nerve injuries
A29	Left side	2 ↷	1	0	0	0	0	0	0	17	..	Specimen lost

* In each case the posterior vertical and horizontal canal was destroyed.

After the operation the dog was kept alive for eighteen days, and careful records kept of his symptoms. He was then anesthetized and killed by having his blood washed out with a solution of Muller's fluid containing 1 per cent. of formaldehyd. The brain and cord were then removed and suspended in Muller's solution, which was changed daily until it no longer became dark. In this way the brain was hardened rapidly and could be cut into thin slabs for immersion in Marchi solution (Muller's solution two parts, osmic acid 1 per cent. one part). We have used this method because we believe it shows nerve pathways with great accuracy, though it requires considerable experience to know how to interpret the findings. One of us (E. S.) obtained such satisfactory results by this method in previous studies on the nervous system that we have used it here. We believe it gives more accurate results than either the developmental method or the secondary atrophy method. The brains were then cut in serial celloidin sections and every third section mounted for study, the intervening sections being kept for reference if necessary. Two points are all important if this method is used: 1. There must be no formaldehyd in the specimen when placed in the Marchi solution, otherwise there will be a fine granular precipitate in the microscopic sections, which is very confusing. 2. Every degenerated nerve must be followed from one section to the other in order to avoid misinterpreting the findings.

A few specimens were hardened in alcohol and then stained with toluidin 1 per cent. for chromatolytic changes.

PURPOSE OF THE INVESTIGATION

In this research we have studied the following points:

1. Do fibers run directly from the ampulla into the medulla?
2. Symptoms produced by destruction of the semicircular canals.
3. The anatomic pathways of the vestibular and acoustic branches of the eighth nerve in the medulla, pons and cerebellum.
4. Symptoms resulting from cutting the eighth nerve.
5. Symptoms resulting from destruction of the nuclei of the eighth nerve.
6. Connections of the three semicircular canals with the eighth nerve nuclei in the medulla.
7. Pathways from the nuclei in which these nerves end to the cerebellum and cerebrum.

This paper does not deal with 5 and 6 as these points require a special series of experiments and will be made the subject of a subsequent paper.

TABLE 2.—SYMPTOMS FROM BONE-WAX TUMORS

No. of Exp.	Bone-Wax Tumors	Nystagmus	Deviation of Eye	Swinging of Head	Attitude	Falling	Rolling	Ataxia	Circus Movements	No. of Days Lived	Stain	Microscopic Findings and Remarks
53	Right	8	0	8	18	0	8	18	8	18	M	Poorly stained. Extensive lesion of cerebellum
59	Left	4	0	0	3	3	0	0	0	18	..	Not examined
64	Left	2	6	0	0	0	0	0	0	18	..	Not examined
66	Right	6	12	12	12	4	4	6	0	18	M	Series incomplete. Extensive lesion involving all of Deiters' nucleus
68	Right	0	21	0	21	0	0	0	0	21	..	Not examined
69	Right	4	12	0	0	0	0	0	11	25	..	Not examined
71	Right	0	14	0	0	8	0	0	0	22	..	Not examined
75	Left	3	3	2	3	0	0	2	0	7	..	Not examined
78	Left	3	3	18	0	0	0	2	0	33	..	Not examined
A32	Left	0	30	0	3	0	0	6	0	32	M	Extensive lesion of lateral lobes of cerebellum extending into the fourth nucleus. Deiters' nucleus and the inferior and middle peduncles have escaped. Very marked degeneration in fillet region
	Cautery to 4th Ventricle											
A1	.....	0	0	0	0	2	0	2	0	5	..	Wound infected; lived six days. Too early for Marchi studies. Symptoms same as in those cases in which inferior peduncle is involved
A11	.....	0	7	3	0	0	0	0	0	18	M	Lesion in posterior longitudinal bundles of both sides. Nucleus IX and inferior peduncle. Lesion too far caudal, too superficial to hit Deiters' nucleus
A21	.....	0	4	0	10	0	0	0	0	18	M	Lesion in lateral cerebellar tract below region of Deiters' nucleus. Marked degeneration going up inferior peduncle

CONNECTIONS OF THE SEMICIRCULAR CANALS WITH THE MEDULLA

After some practice, one of us (B. Y. A.) perfected his technic so that it was possible for him to destroy either the posterior vertical canal or the lateral canal. It is not possible, however, to make an isolated lesion of the anterior vertical canal, and in order to study the result of the destruction of this canal all three canals were destroyed, and thus by a process of exclusion it was hoped to determine the course of the fibers supplying this canal. It was our hope that by studying the chromatolytic changes in the nuclei we might discover whether certain nuclei supplied certain canals. Our results along this line, however, were not convincing, and this phase of the problem still remains to be solved. We propose to attack the problem next by studying the secondary or indirect Wallerian degeneration and hope in this way to get some answer.

It has seemed probable to us that since there are three nuclei that compose the vestibular group, the nucleus of Deiters, nucleus of Bechterew and nucleus triangularis, each one of these is connected with one semicircular canal. If the various pathways that the Bárány followers use to explain the symptoms resulting from the stimulation of one canal are to be accepted, the connection of these nuclei with the cerebellum must first be worked out. Up to the present time there is insufficient anatomic basis for their deductions.

One of the disputed points about the canals has been whether after destruction of an ampulla degenerated fibers could be traced up the vestibular nerve. Tricomi-Allegra,[18] Matte [19] and Weigner [16] all claimed to have found such fibers, but a careful repetition of the work by Van Gehuchten [17] and his pupil Michotte do not bear this out. Our specimens bear out the observations of Van Gehuchten. In no case have we found degenerated fibers passing up the vestibular nerve after destruction of the semicircular canals.

We believe, therefore, that all fibers from the canals end in Scarpa's ganglion and that a second neuron carries impulses from here to the vestibular nuclei.

SYMPTOMS ARISING FROM DESTRUCTION OF THE SEMICIRCULAR CANALS

Practically all investigators have described a series of symptoms as characteristic of destruction of the semicircular canals. Wilson and Pike [19] enumerate them thus: "Deviation of one eye downward and outward, nystagmus with the slow phase to the injured side, inability to walk, falling to the injured side, head inclined to one side, falling to the injured side and going continually in a circle. At the end of the second week the animal stood erect with torsion of the head to one side and body inclined to one side."

TABLE 3.—RESULTS OF CUTTING EIGHTH NERVE

No. of Exp.	Operation	Nystagmus	Deviation of Eye	Swinging of Head	Attitude	Falling	Rolling	Ataxia	Circus Movements	No. of Days Lived	Stain	Microscopic Findings and Remarks
77	Right eighth nerve cut	4 ↖	18	7	7	4	0	7	0	18	M	Bilateral lesion of paraflocculus and flocculus. Small hemorrhage into upper portion of Deiters' nucleus. A few fibers to nucleus tecti, some fibers to posterior longitudinal bundle. Arcuate fibers to posterior corpora quadrigemina. Descending fibers to cord. Right eighth nerve cut
89	Right eighth nerve cut	0	0	0	0	0	0	0	0	9	..	No microscopic confirmation of operation. Too early for Marchi staining.
90	Right eighth nerve cut	0	0	0	0	0	0	0	0	18	M	Lesion mostly of vestibular portion. Slight lesion of flocculus. Fibers to nucleus tecti, few fibers to posterior longitudinal bundle. Arcuate fibers. No fibers to posterior corpora quadrigemina. Some fibers to cord. Right eighth nerve cut
92	Right eighth nerve cut	4 ↖	0	0	0	0	0	2	0	18	M	Small lesion of posterior dorsal and postventral paraflocculus and eighth nerve. A few fibers seen in posterior longitudinal bundle going as far forward as fourth nucleus; none to third. Fibers to Deiters', arcuate, posterior corpora quadrigemina and cord
93	Right eighth nerve cut	6 ↖	8	0	0	0	0	0	0	18	M	Small lesion of eighth and paraflocculus. Fibers to Deiters' to opposite corpora quadrigemina nucleus tecti, arcuate fibers and to cord. Fibers to posterior longitudinal bundle, but stop before they get near the fourth nucleus
94	Right eighth nerve cut	2 ↑	0	0	0	0	0	0	0	18	M	Specimen poorly stained. Eighth nerve cut but study of tracts not possible
100	Right eighth nerve cut	5 →	0	5	5	5	5	5	0	5	..	Injury to middle peduncle. Animal died on fifth day, too early for Marchi staining. These animals often die from exhaustion due to thrashing around. No other cause of death. Right eighth nerve cut
101	Right eighth nerve cut	3 →	0	0	0	0	0	0	0	3	..	Died too soon after operation for Marchi staining. Absence of symptoms indicates cerebellar nuclei and peduncles were not injured. Right eighth nerve cut
102	Right eighth nerve cut	3 ↖	3	0	0	0	3	0	0	3	..	Died too early for Marchi stain. Some evidence of injury to peduncle stained with thionin. Eighth nerve cut
108	Right eighth nerve cut	0	0	0	0	0	0	0	0	18	M	Eighth nerve not cut. Injury to flocculus and paraflocculus. Lesion did not extend into cerebellar nuclei. No degeneration except to cerebellar nuclei
116	Right eighth nerve cut	0	0	0	0	0	0	0	0	19	M	Injury to flocculus and paraflocculus lobules. Extensive degeneration to cerebellar nuclei, but no involvement of Deiters' or cerebellar nuclei. Some degenerated fibers in posterior longitudinal bundle in region of fourth nucleus. Eighth nerve cut
118	Left eighth nerve cut	3	3	3	3	3	3	3	3	3	..	Eighth nerve cut and inferior peduncle injured. Died too soon for Marchi staining. Died, apparently, from exhaustion as result of violent thrashing around
130	Left eighth nerve cut	3 ←	3	0	0	0	0	0	0	18	M	Partial lesion of eighth nerve, chiefly of cochlear portion. A few fibers to Deiters' nucleus; none to posterior longitudinal bundle; arcuate fibers and some to cord. A small bone-wax tumor present which produced a marked flocculus lesion

133	Right eighth nerve cut	1 →	1	0	0	0	0	1	0	1	..	Right eighth nerve cut. No evidence of other lesion in pons or Deiers' nucleus. Too early for Marchi staining. Death due to hemorrhage
138	On canals	1 ⇝	0	0	10	0	0	2	0	14	M	This case originally belonged to Table 1, but the sections showed that in removing the entire otic bone the eighth nerve had been injured. Fibers to nucleus tecti, corpora quadrigemina of opposite side; peduncle and cerebellum uninjured. No fibers in posterior longitudinal bundle running up to sixth or fourth nuclei
A3	Right eighth nerve cut	8 ↘	10	2	10	0	0	0	0	18	M	Eighth and seventh nerves cut. Injury to flocculus and paraflocculus lobes with involvement of cerebellar nuclei; diffuse lesion not well localized; of value for the symptoms rather than the anatomic tracts
A5	Right eighth nerve cut	6 ↘	10	2	18	0	0	6	0	18	M	Lesion of Deiters' nucleus as well as eighth nerve. Fibers pass to anterolateral and contralateral portion of cord. Fibers to posterior longitudinal bundle, but none run forward to sixth and fourth nuclei. Fibers to corpora quadrigemina of opposite side and to cord
A8	Right eighth nerve cut	2 ↘	3	7	9	9	9	9	0	13	..	Wound infection. Eighth nerve cut. Rather extensive trauma to cerebellum. Too early for Marchi symptoms like those seen when peduncle and cerebellar nuclei are involved
A15	Right eighth nerve cut	5 ↘	5	5	5	5	0	0	3	5	..	Died of distemper; wound healed. Eighth nerve cut. Extensive trauma to cerebellum. Too early for Marchi staining. Symptoms like those seen in lesions of peduncles
A25	Right eighth nerve cut	2 ↘	2	0	0	0	0	2	2	2	..	Too early for Marchi staining. Eighth nerve cut. Large blood clot pressing on lateral lobe of cerebellum. Symptoms like those seen when cerebellar nuclei involved
135	Right eighth nerve cut	0	0	0	0	0	0	3	0	18	M	Eighth nerve cut, anterior and post paraflocculus slightly injured, no involvement of cerebellar nuclei. Fibers to Deiters' nucleus, nucleus tecti and corpora quadrigemina of opposite side. Fibers to posterior longitudinal bundle, but none to fourth and sixth nuclei. Some fibers to cord
136	Right eighth nerve cut	4 ↘	3	3	0	0	0	7	0	18	M	Eighth nerve cut. Hemorrhage into Deiters' nucleus. Poor Marchi penetration. Symptoms characteristic of lesion in region of Deiters' nucleus
A16	Right eighth nerve cut	5 ↘	16	4	6	0	0	10	0	18	M	Eighth nerve cut. Lesion of paraflocculus anterior and posterior quite extensive. Two microscopic hemorrhages in Deiters' region. Symptoms characteristic of Deiters' lesion. Fibers as in other cases of Deiters' lesions. Fibers to posterior longitudinal bundle; none go forward to fourth and sixth. Some fibers end in nucleus globosus and embolus in cerebellum
A18	Right eighth nerve cut	3 ↘	14	3	8	0	1	8	0	18	M	Infected hematoma. Eighth nerve cut. Small abscess extended into Deiters' region. Symptoms as in other Deiters' cases. Is confirmatory but in itself not a good case because of infection. Fibers as in other cases of Deiters' lesions. Fibers to posterior longitudinal bundle stop before they reach fourth and sixth nuclei
A26	Right eighth nerve cut	3 ↘	7	7	85	0	1	7	0	85	M	Eighth nerve cut. Lesion of Deiters' nucleus and inferior peduncle and anterior and posterior paraflocculus. Fibers in posterior longitudinal bundle and corpora quadrigemina and nuclei tecti. A few fibers traced to region of descending root of fifth nucleus around aqueduct of Sylvius
A33	Left eighth nerve cut	3	16	0	3	0	0	3	0	31	M	Pure lesion of eighth nerve. Fibers go to Deiters' nucleus and stop there; a few arcuate fibers. Nothing found histologically to explain ataxia or attitude of head

We have observed these symptoms in the eight cases of bone-wax tumor and also in cases in which we attempted to cut the eighth nerve intracranially, but in every case in which other symptoms than nystagmus or deviation of the eye occurred there was an injury either to the nuclei of the cerebellum or peduncles of the pons. In fact, it is possible by looking over Table 1 to pick out from the symptoms those cases in which there were other lesions.

These symptoms, as we have observed them, may be grouped under eight headings:

1. *Nystagmus.*—In every case in our series in which this phenomenon has been observed it has been bilateral, of the so-called labyrinthine type, that is, consisting of a slow movement of the eyeball in one direction and a quick movement in the opposite direction. In some instances the motion was purely lateral or vertical and in others it was definitely rotatory in character; this we speak of as clockwise and anticlockwise motion.

We have seen a definite relation between the side of the lesion and the direction of the nystagmus. In all left-sided lesions the slow component was toward the left side where purely horizontal motion was observed; and the slow phase was in the clockwise direction, that is, the top of the eye moved toward the animal's left when rotatory nystagmus occurred. With right sided lesions, the opposite was observed.

Nystagmus was the most frequently observed symptom. It was usually seen as soon as the animal recovered from the anesthetic, when it was in some cases very rapid, and in most cases it had entirely disappeared within seventy-two hours, often within twelve hours, though it sometimes persisted for four or five days.

2. *Deviation of Eye on Side Corresponding to Operation.*—The deviation was usually downward and outward. In rare instances downward and inward. Occasionally the eye on the opposite side showed some deviation downward, but in all animals of this series this symptom has been rarely observed.

The degree of deviation is markedly influenced by the position of the animal's head, frequently being unnoticed when the animal is at rest in the attitude of choice. It is usually increased by pointing the snout upward or by turning the head in such a way that the operated side is uppermost. This is the most persistent symptom observed in the entire study, usually remaining unchanged throughout the eighteen days during which the animal was kept alive after operation.

3. *Swinging of Head.*—This striking symptom was observed only when the animal was held up by grasping it about the thorax and holding it in such a manner that the snout pointed almost vertically. The animal would then swing its head about in a circle, first bending its neck

toward the operated side, then backward, then toward the opposite side, then forward and then to the starting point. This movement in well marked cases was carried out with great rapidity and violence and kept up till either the animal or the observer was exhausted. The tendency to swing the head thus was soon lost and usually was not observed for more than three or four days.

It was not observed in any animal with a simple labyrinthine lesion.

4. *Attitude of Head.*—The head is rotated about its long axis in such a way that the injured ear points to the corresponding shoulder. In some more marked cases the neck is flexed toward this side, sometimes to 90 degrees or more. This peculiar attitude did not always become apparent in the first twenty-four to forty-eight hours and usually persisted till the animal's death, though it gradually diminished and possibly would have entirely disappeared had the animals been kept alive for a much longer time.

5. *Falling toward the Operated Side.*—This symptom apparently was due to a weakness in the legs on the side corresponding to the lesion and to spasticity of the opposite extremities. When this symptom occurred it usually persisted only for from twenty-four to seventy-two hours after operation.

6. *Rolling about Long Axis of Body.*—The direction of the rolling is dependent on the side of operation. Starting from the normal resting position the animal rolls over and over toward the operated side, finally coming to rest with the operated side downward.

7. *Ataxia.*—This was observed when the animal was walking about.

8. *Circus Movements.*—Animals exhibiting this symptom had a tendency to go round in circles as about a ring and were unable to proceed by a direct course from one point to another. The accompanying tables show the number of animals of this series exhibiting the various symptoms described.

In pure lesions of the semicircular canals, however, the only symptoms we observed were nystagmus and occasionally deviation of the eye on the operated side. The type of nystagmus was not always the same when a certain canal was destroyed; thus, in Experiments 124 and A31 there was clockwise nystagmus when the left horizontal canal was destroyed, while in Experiments 56 and 132 it was lateral. The same variation in symptoms was observed when two canals were destroyed, but it was striking that whereas nystagmus occurred in all cases in which one canal was destroyed, it often was absent when two canals were destroyed. For this observation we have no explanation to offer, but merely record the fact. The same symptoms were observed when all three canals were destroyed. In no case did the nystagmus last more than two days.

From these experiments we feel justified in concluding that in all previous experiments the varied symptoms ascribed to destruction of the canals must have been due to intracranial injuries which could only have been discovered on microscopic studies. Our contention is supported by the experiments on the eighth nerve for in those cases also no such symptoms as are described by Wilson and Pike [19] and others were observed unless there were other injuries of the nervous system. A small intracranial hematoma in some of our cases was enough to produce these symptoms.

Baginsky,[5] thirty-five years ago, in referring to von Bechterew's work, pointed out that the symptoms were due to an injury of other regions.

ANATOMIC CONNECTIONS OF VESTIBULAR AND ACOUSTIC BRANCHES OF THE EIGHTH NERVE IN THE MEDULLA, PONS AND CEREBELLUM

Whereas the cochlear branch of the eighth nerve has frequently been studied, the vestibular portion has been sadly neglected. In going over the literature, we have been impressed with the varied descriptions of the nuclei of the vestibular nerve. Our experiences bear out, however, the statements of other observers that the fibers pass to the three nuclei, Deiters' (Fig. 1), Bechterew's and nucleus triangularis. In a personal communication with Dr. Elliot Smith of University College, London, he told us that he has been teaching his students that on account of the confusion existing in regard to the vestibular nuclei he has applied the term Deiters' nucleus to the entire group of cells. This we are not willing to do as yet as there are such varied types of ganglion cells in these nuclei that we hope to be able to show that they have different functions.

Our specimens show fibers passing from the vestibular nerve to the nucleus tecti, (Fig. 8) as claimed by Cajal.[24] Our specimens also show degenerated fibers which pass from the paraflocculus lobe (Elliot Smith [25]) to the nuclei of the cerebellum (Fig. 4). The degeneration appears in all of our specimens and is due to the slight pressure on the paraflocculus while retracting it to expose the eighth nerve. As we have seen no fibers passing beyond the vestibular nuclei, we believe that redistribution of the fibers takes place after they end in these nuclei. This brings us to the neurons passing from the vestibular nuclei upward.

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24. Cajal: Beitr. zum Studium der medulla oblongata, Leipzig, 1896, p. 75-101. Die Endigungen des Aüserem Lemniscus, oder die sekundäre akustische Nervenbahn, Deutsch. med. Wchnschr. **28**:275, 1902. Les ganglions termineaux du nerf acoustique des oiseaux, Jour. Psychol. u. Neurol. **13**:214-239, 1908.

25. Smith, G. E.: The Primary Subdivision of the Mammalian Cerebellum, J. Anat. & Physiol. **36**:375. Notes on the Morphology of the Cerebellum, J. Anat. & Physiol. **17**:329, 1902-1903.

In our series we have six lesions of Deiters'[26] nucleus (confirmed microscopically)—Experiments 77, 136, A5, A16, A18 and A26. These have varied in size; thus A16 had only two-minute hemorrhages. In addition the animals in Experiments 53, 66, 75, 78, 100, 102, 118, A3, A8, A15 and A25 had the same symptoms that the patients with confirmed Deiters' nucleus lesions had. In some of these cases, as shown in the tables, histologic studies were made and showed extensive lesions of the cerebellar nuclei as in Experiments A3, A8, A15, and it is fair to suppose that all these animals had a lesion either in Deiters' nucleus, in the cerebellar nuclei, or in the pathways connecting the two. The symptoms did not enable us to differentiate lesions in these various regions. It should be mentioned that the late Victor Horsley and R. H. Clarke made some elaborate studies about fourteen years ago on the anatomy and physiology of the cerebellar nuclei, but unfortunately these were never published. Their studies contained evidence which might have enabled one to differentiate lesions in these different regions.

A21 and A1 are of great interest, however, in that in these animals the lesion lay in the pons but caudal to Deiters' nucleus, and in these cases the symptoms were few. This indicates that destruction of the fibers running cephalad from Deiters' neucleus are responsible for those symptoms about which there has been so much discussion. This observation is in accord with those of Fraser.[15] In none of our lesions were all the cells of the acoustic portion of the eighth nerve destroyed. In all cases we found fibers passing into the cord, as described by Fraser and Lloyd[27] and Russell[28] and Mott.[29] These fibers pass to the anterolateral portion of the cord on the same side, and a few cross over to the anterolateral region of the contralateral side. A few fibers were seen passing to the posterior longitudinal bundle of the same and opposite sides (Fig. 7). These ended just before reaching the region of the fourth nerve, and none was seen to go further forward (Fig. 8). We have been unable to confirm the observations of Fraser that large numbers of fibers pass up the posterior longitudinal bundle. In none of our cases, however, were the lesions as large or extensive as those that Fraser produced, which are illustrated in his paper.

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26. The nucleus which bears Deiters' name was so named by his teacher, Max Schultze, who edited Deiters' work, as Deiter died very suddenly as a young man before completing his work. According to the illustration which Schultze published, and which we reproduce, it seems to us that the portion of the nucleus is that to which Bechterew later attached his own name.

27. Lloyd, R. E.: On Chromatolysis in Deiters' Nucleus After Hemisection of the Cord, J. Physiol. **25**:191, 1900.

28. Russell, Risien: The Origin and Destination of Certain Afferent and Efferent Tracts in the Medulla Oblongata, Brain **20**:409, 1897.

29. Mott, F. W.: Experimental Enquiry upon the Afferent Tracts of the Central Nervous System of the Monkey, Brain **18**:1, 1895.

It is of great importance to note that these fibers lie close to the floor of the fourth ventricle and continue to lie so superficially as they go cephalad that any pressure, such as from an internal hydrocephalus, may readily affect them. We have had several patients with hydrocephalus without other lesions, as shown by necropsy, in whom the Bárány tests showed abnormalities. We believe that the internal hydrocephalus associated with almost all posterior fossa lesions is responsible for the abnormalities observed in the Bárány tests.

A large group of fibers are seen passing up to the cerebellum and seem to end in the nucleus tecti (Fig. 8). We have not seen any fibers pass to the lateral lobes of the cerebellum either of the same or opposite side. Our results do not show any connection with the lateral lobes of the cerebellum. Dr. Elliot Smith confirms this observation, and he has informed us that observations by Gordon Holmes (as yet unpublished) on a large number of gunshot wounds of the cerebellum, have shown no demonstrable disturbances of the arms, legs, or trunk as Bárány asserts occur when there are lesions of certain portions of the lateral lobes.

We have found arcuate fibers running across the median line (Fig. 7) ending in the superior olive, as most other observers have found. A few pass on into the lateral fillet and end in the inferior portion of the posterior corpus quadrigeminum of the opposite side (Fig. 9). Van Gehuchten asserted that these fibers end before they reach the corpus quadrigeminum, but we have traced them directly to that region. We have found no fibers that run more anteriorly and have found no fibers that go to the external geniculate body.

SYMPTOMS RESULTING FROM CUTTING THE EIGHTH NERVE

In the twenty-seven experiments in which we cut the eighth nerve a large number have had to be considered under a different heading since more was destroyed than merely the eighth nerve. These cases can be identified almost as accurately by the symptoms as by a study of the anatomic specimens. The cases that appear at the end of Table 3, in which the animals showed swinging of the head, circus movements, etc., all had lesions of other regions in addition to destruction of the eighth nerve.

The only symptoms presented by patients with pure eighth nerve lesions were deviation of the eye downward and outward, nystagmus with the slow phase anticlockwise and in three cases, Experiments 92, 135 and A26, a transient ataxia in walking which was so slight that we had some doubt as to its existence. We thought at first the inevitable slight injury to the paraflocculus might be responsible for this ataxia, but a more careful analysis of our cases and the total absence of this symptom in certain cases of injury to the paraflocculus definitely contradicts that idea.

One clinical case supports these experimental findings. On this patient the eighth nerve was cut for distressing tinnitus. His symptoms were lateral nystagmus to either side and slight ataxia for a few days. These symptoms are similar to our experimental results. Certainly, cutting the eighth nerve in man produces none of the symptoms that have been reported by others when the eighth nerve is cut in animals.

In seventeen experiments the lesion was in the region of Deiters' nucleus. These animals all showed:

1. Deviation of the right eye and facial paralysis.
2. Rotatory nystagmus counterclockwise.
3. Swinging of the head.
4. Inability to stand.
5. Marked ataxia.

Some animals showed the symptoms for longer periods than others. When the injury was not so severe there was a certain amount of readjustment apparently, while in others there was no evidence of this. We consider it important to disregard symptoms that are present only a few hours after operation. There must be a certain degree of permanency in order to consider them of value. Some observers have described symptoms that were noted when the animals came out of the anesthesia; this we have not done.

The symptoms we have noted are those described by von Bechterew and others as characteristic of an eighth nerve lesion, but we believe we have shown that the symptoms of rotation often called circus movements are not produced by cutting the eighth nerve but are due to injury to neighboring structures, especially the middle and superior peduncles of the cerebellum. The so-called cerebellar attitude of the head seen in these animals is due to injury of the middle peduncle and not, we believe, a protective mechanism as has been claimed by Cushing. That the cerebellar attitude is due to involvement of the peduncle was shown by Victor Horsley more than ten years ago.

In all our lesions of the eighth nerve we have noted deviation of the eye down and out. We are unable to find a satisfactory explanation for this.

The great difference between our results and those of others we believe to be due to the fact that they operated in such a manner as to be unable to see exactly what they did, and as careful histologic studies were not made, at least are not recorded in any of these articles, the injuries responsible for these symptoms were overlooked.

SYMPTOMS ARISING FROM BONE-WAX TUMORS

For the sake of completeness we add a few remarks about this group of cases. The symptoms which these animals presented are for the most part the same as those presented by our animals with Deiters'

nucleus lesions. Unfortunately, as stated previously, most of these cases were not studied histologically but in Expriment A32 such studies were made. The sections of this animal are very instructive for, while the lesion of the cerebellum is extensive and extended into the cerebellar nuclei, the inferior peduncle and middle peduncle escaped injury. The severe symptoms of rolling and circus movements seem to be directly dependent on injury of the inferior peduncle. This was the case in Experiments 77, 100, 118, A8, A15 and A26.

CONCLUSIONS

1. There are no fibers running directly from the semicircular canals to the nuclei of the vestibular nerve.

2. The fibers running from Deiters' nucleus anteriorly in the posterior longitudinal bundle are few in number and stop before they get to the third and fourth nuclei so that it is doubtful whether there is any connection between Deiters' nucleus and the other nuclei. This is the oculovestibular tract spoken of by Wilson and Pike.

3. We have found no fibers passing from Deiters' nucleus to the lateral lobes of the cerebellum where the centers Bárány has described lie.

4. All anatomic evidence to support Bárány's contention as to the connections between Deiters' nucleus and the cerebellum is lacking.

5. There are fibers from Deiters' nucleus that end in the posterior corpus quadrigeminum of the opposite side, but no neuron goes directly to the external geniculate.

6. All fibers of the vestibular nerve end in one of the three vestibular nuclei or in the nucleus tecti of the vermis.

7. Circus movements, rolling over and over, ataxia, swinging of the head, attitude of the head, hitherto described as due to a lesion of the eighth nerve or destruction of the semicircular canals, are due to injuries to the cerebellar nuclei or middle peduncle.

8. The so-called cerebellar attitude of the head is due to involvement of the middle peduncle.

9. Nystagmus and deviation of the eye downward and outward are the only constant symptoms observed after pure lesions of either the semicircular canals or eighth nerve.

10. As the vestibular fibers lie so near the floor of the fourth ventricle, the symptoms described by Bárány as due to isolated lesions in the pons may be produced by, and probably usually are due to, the internal hydrocephalus which is a common accompaniment of posterior fossa lesions.[30]

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A STUDY OF THE CORTICAL OLFACTORY CENTER

BASED ON TWO CASES OF UNILATERAL INVOLVEMENT OF THE OLFACTORY LOBE

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It is many years since Broca and Zuckerkandl first claimed that the gyrus hippocampi, cornu ammonis, and gyrus fornicatus were the cortical olfactory centers. Experimental study, clinicopathologic observations, and comparative anatomy have been productive of a large number of papers concerning this important subject. In spite of the efforts of many authors, the exact location of the cortical olfactory center cannot be said to have been definitely determined. Recently I had an opportunity to study two patients presenting unilateral involvement of the olfactory lobe, one of congenital absence, the other of softening due to arteriosclerosis. Both had a lesion in the same area of the homolateral lobus pyriformis, the lesion being reasonably regarded as a secondary degeneration following the primary involvement of the peripheral olfactory center.

REPORT OF CASES

CASE 1.—*Clinical History.*—Case No. 22048, Danvers State Hospital; necropsy No. 2165. A man, born in August, 1861, single, whose early life and development were normal, and who had received a common school, possibly a high school, education; who had worked as a letter collector; when about 25 years of age had had some form of convulsions, followed by several others in the course of a few years. Two years previous to admission he had the first attack in a number of years. While working in May, 1919, he fell and was carried home unconscious. On Jan. 2, 1920, he had another attack and was taken to the hospital. Becoming somewhat restless and disturbed, especially at night, he was transferred to the Danvers State Hospital.

Examination.—He was fairly well developed but poorly nourished. His chest was somewhat paralytic; respiratory movements were regular. Throughout both sides of the chest were a few scattered râles. Heart sounds were weak and regular. The pulse rate was 75 per minute; good quality. The blood pressure was systolic 140, diastolic 90. Nothing unusual was noted on palpation of the abdomen. The urine was normal; the Wassermann reaction was negative.

At times he complained of attacks of vertigo. The conjunctiva was somewhat infused; the pupils were irregular and reacted promptly to light and accommodation. The visual field was somewhat restricted. On account of the poor cooperation of the patient, the sense of taste and of smell could not be examined. The knee jerks were somewhat increased. Superficial reflexes were

apparently retained. The facial muscles showed some lack of motility. There was considerable loss of power in both hands. The Romberg sign was positive. There was gross tremor on exertion.

Course of Disease.—In the ward he was talkative, elated, restless, disoriented and more or less confused. This condition persisted for two or three days when he became dull, depressed, stupid and apathetic. He gradually began to lose interest in things about him, and it was with great difficulty that he could be made to take sufficient nourishment. He remained practically in the same condition until Jan. 22, when he died, the cause of death being bronchopneumonia.

Postmortem Examination.—Necropsy was performed eight hours postmortem. Anatomic diagnoses were: emaciation, cyanosis of fingers, phimosis, chronic adhesive pleuritis, bronchopneumonia, hypertrophy of left ventricle, sclerosis of aorta, chronic passive congestion of liver, chronic diffuse nephritis, hypertrophy of prostate, chronic pachymeningitis and leptomeningitis, slight atrophy of central and frontal convolutions and absence of the right olfactory lobe.

Head and Brain.—The scalp was thin, and adherent in the frontal region. The calvarium was thick and dense, diploë being absent. The internal table was eroded along the longitudinal sinus. The groovings for the middle meningeal arteries were rather faintly marked. The dura mater showed marked thickening, and was slightly adherent to the calvarium. The pia mater was thickened and clouded over the vertex, especially in the frontal and central regions. Moderate subpial edema was noted in these regions. The convolutions of both hemispheres were slightly atrophic, the sulci being somewhat widened. The carotids, basilar and major arteries of the cerebrum and cerebellum were only slightly thickened. No atheromatous patches were noted.

The right olfactory bulb with its peduncle was missing. At the root of the olfactory sulcus a small elevation about the size of the head of a pin was found, corresponding to the olfactory trigon. On the right side the stria olfactoria, medialis and lateralis could not be identified, while on the left were well marked whitish striations. Substantia perforata anterior of the right side was markedly deeper than that of the left. Lobus pyriformis, gyrus hippocampi, cornu ammonis, and gyrus fornicatus were alike on both sides and did not show any malformations or agenesis. A difference of the convolutional pattern was noted in the frontal poles. Sulcus frontomarginalis of Wernicke was prominent on the right, while on the left it was rudimentary. Otherwise there was no gross difference in the hemispheres, which were equal in weight (150 gm. after fixation). Frontal section, made after Meynert, revealed nothing remarkable in any of the planes. The fossae of the cribriform plates, where the olfactory lobes rested, showed an abnormal condition corresponding to the unilateral absence of the olfactory bulb. The left side was wide and flat, as in a normal person; the right side was very shallow with little space for the olfactory bulb.

Nothing unusual was noted in the pituitary body, the pons, cerebellum and medulla.

The spinal cord showed nothing of note.

Microscopic Examination.—Practically all parts of the lobus pyriformis, gyrus hippocampi, and cornu ammonis were submitted to special investigation. Several blocks from the gyrus cinguli and isthmus of the gyrus fornicatus were carefully studied, as well as other important areas of the cerebral cortex. Thionin staining, Weigert-Pal's method for staining myelin sheaths, Mallory's method for neuroglia demonstration, Herxheimer's scharlach r staining, and several other general staining methods were employed.

Outside the rhinencephalon, the microscopic examination showed only minor alterations. The pia mater over the vertex was thickened, but there was no perivascular cellular exudate. In the frontal area, precentral and postcentral convolutions, was a small number of more or less degenerated ganglion cells, the stratigraphical picture not being much altered. Neuroglia cells and fibers were noticeably increased generally. Marginal neuroglia fiber nets were

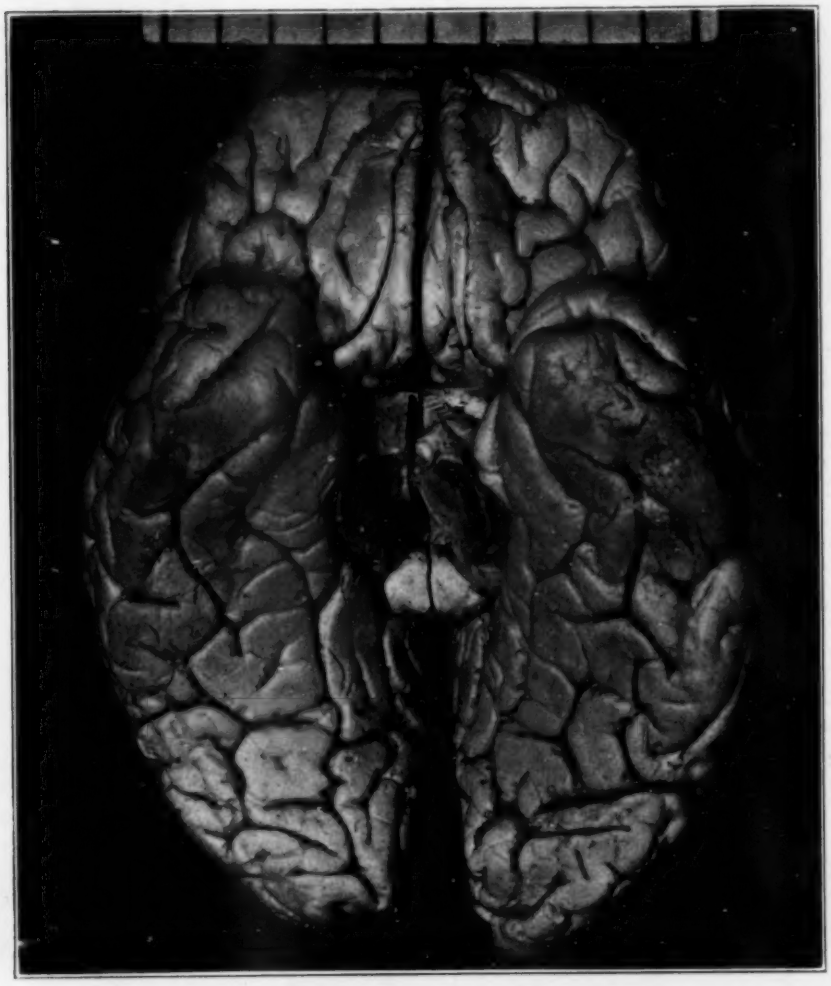

Fig. 1.—Brain without the right olfactory lobe.

thickened. A well marked envelop formation was observed around the large cortical vessels. The myelin sheaths showed no marked involvement. In the entire limbic lobe of the right side, the most marked alteration was found in the lobus pyriformis, lateral to the gyrus circumambiens and anterior to the uncus proper.

Before taking up the pathologic findings, I shall give in brief the normal histology.[1] As is known to histopathologists, the lobus pyriformis presents a large number of peculiar cell nests in the small pyramidal cell layer, giving a bizarre appearance to the cortex. The cell nests consist of islets of small pyramidal cells and islets of giant polymorphonuclear cells, the latter being more prominent. Cajal[2] attributes an important olfactory function to the area over which the peculiar cell islets are distributed. In place of medium and large sized pyramidal cells there are pyramidal cells of equal size with more or less deviated apical prolongations. Below this is a narrow layer standing out on account of the sparsity of nerve cells. Still deeper we come to the large and small fusiform layer, as in the other cortex. The zonal layer is well developed and composed of dense fibers. Corresponding to the islets of cells described, circular areas sparsely supplied with fibers are found. The line of Baillarger is wide but its fibers are sparse.

The clusters of large polymorphous cells had for the most part disappeared, showing only a few scattered cells which were no longer in the formation of nests. The cytoplasm, as well as the nucleus, was much shrunken. The body plasma stained uniformly dark and Nissl bodies were barely visible. The nuclei were irregular in outline and located in the periphery of the cell body, either toward the apex or the side of the cell. The clusters of small pyramidal cells were equally involved, and the cells were difficult to identify. In consequence of the above condition the lamina, containing the characteristic cell clusters, often tended to show blank areas almost bare of nerve cells. The number of cells in the following layer were noticeably decreased; this frequently showed areas devoid of ganglion cells. Many cells were chromophilous and shrunken, their dendrites being traced for a considerable distance; a small number, however, were stained pale and presented a shadow-like appearance (Zellschattenfigur). The large and small fusiform cells of the deeper layer were uniformly shrunken with a few advanced forms of degeneration.

The tangential fibers in the plexiform layer were greatly involved, containing many degenerated fibers and a few swollen elements. Corresponding to the layers of cell clusters and underlying cells, the myelin sheaths showed the most marked degeneration. Radial, as well as inter-radial fibers had undergone degeneration, leaving pale stained areas. The fibers in the line of Baillarger were barely identified. The medulla was somewhat paler than normal with apparently degenerated fibers.

An extreme fibrillar gliosis was found in the plexiform layer, in the lamina of cell clusters, and in the upper part of the pyramidal cell layer. Fine fibers were densely interwoven throughout these layers, showing no definite demarcation between these and the neuroglia network of the cell-free border. In the deeper cell layers and in the upper part of the white matter were numerous spider-cells with luxuriant fibrillation, the fibrils being of rather small caliber.

The whole picture suggested a chronic long-standing process, apparently the result of the nonfunctioning of this part of the brain. It must be emphasized that no marked alterations of any kind were found in the same area of the contralateral hemisphere of the brain.

In the cornu ammonis, when compared with the healthy side, slight cell sparsity was found in the stratum cellularum pyramidalium and the terminal

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1. Campbell: Histologic Studies on Localization of Cerebral Function, New York, G. P. Putnam's Sons, 1905.

2. Cajal: Histologie du systeme nerveux de l'homme et des vertèbres. Ed. 2, Paris, 1911.

plate. The cells showed well marked fatty pigmentous deposits, mostly in the apical prolongations. However, such deposits were found in cells of the healthy side. Myelin sheaths showed no marked degeneration. The neuroglia elements were not remarkable. No decided alterations were demonstrated in the rest of the limbic lobe.

COMMENT ON CASE 1

In the literature, cases showing the absence of the olfactory bulb are rare. Cases were reported by Bernard,[3] 1858; Kundrat,[4] 1882; Duval,[5] 1884; Valenti,[6] 1911; Ranke,[7] 1913; Weidenreich,[8] 1914; Haga,[9] 1915, and Tanaka,[10] 1917. With the exception of Tanaka's patient, no one of these was subjected to a research investigation of the supposed center of the olfactory sense. Tanaka found most marked alterations in the cornu ammonis and less marked, but fairly well-pronounced, changes in the hippocampal gyrus proper. He avoided the conclusion, however, that the changes found were due to the secondary degeneration following the primary arrest of development of the olfactory lobes—since his patient was an epileptic, and the same changes would be found in epileptic brains without involvement of the olfactory apparatus. It is interesting to know that the present patient also showed epileptic convulsions in earlier life. (This will be referred to later in the comment on Case 2).

Weidenreich's patient, although deprived of both olfactory lobes, was said to have possessed some olfactory sense during life. The olfactory nerves must have taken some abnormal course into the cortical center. It is to be regretted that in the present patient the olfactory sense was not tested, as in Tanaka's case. The question may arise, particularly in this case, as to whether or not the cortical olfactory center developed normally, also whether the nerve tract ran an abnormal course to the peripheral olfactory apparatus. To this I can give no definite answer, but the long-standing degenerative process, found in

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3. Bernard, Claude: Leçons sur la physiologie et la pathologie du système nerveux **2**:1858.

4. Kundrat: Arhinencephalie, 1882; abstr. in Schwalbe: Die Missbildungen des Kopfes, 1913.

5. Duval: Bull. Soc. d'Anthropol. de Paris, Ed. 3 **8**:1884.

6. Valenti: Un cas d'absence unilaterale de l'appareil olfactif, Arch. ital. de biol. **64**:457, 1911.

7. Ranke: Zytoarchitektonik der Grosshirnrinde in einem Fall von Kyklopie, 1913; Referat in Folia neurobiologica, 1913.

8. Weidenreich: Ueber partiellen Riechlappendefekt und Eunuchidismus beim Menschen, Ztschr. f. Morphol. u. Anthropol., 1914.

9. Haga: Eenzijdig ontbreken van den tractus olfactorius, Nederl. Tijdschr. v. Geneesk. Jaargang 1915, No. 22, pp. 2450-2452.

10. Tanaka: Absence of Lobus Olfactorius and Sclerosis of Cornu Ammonis, Arch. Neurol. & Phychiat. **3**:151 (Aug.) 1920.

the supposed cortical olfactory area, would suggest the nonfunctioning of the unilateral olfactory tract.

The cause of the defect of the olfactory lobe is not known. Ernst [11] holds the compression theory, while Schwalbe [12] attributes this condition to embryonal defect. Weidenreich interpreted his case as resulting from the failure to combine the olfactory nerve and the olfactory lobe, in consequence of which the latter tends to atrophy. In the present case the cribriform plate of the right side showed some deformity. This condition would probably suggest the difficulty which had existed in effecting the combination of central and peripheral olfactory organs, which, according to His, occurs in a relatively later embryonal stage.

Case 2.—*Clinical History.*—Case No. 22265, Danvers State Hospital; necropsy No. 2202. A man, born in Kittery, Me., March, 1863, married and the father of six children, had a negative family history for mental disease. Little was known about his early life and development. He was a general laborer, used alcohol to some extent, and was a heavy smoker. The onset of the present psychosis began following a "shock," when he became stupid and dull. From that time he had had periods of marked confusion, occurring about once a month and simulating epileptiform seizures. More recently he suffered a more severe attack, when he remained in bed four days, and showed a marked loss of memory. It was impossible to hold his attention for any length of time on any subject, and he showed a tendency to irrelevance and desultoriness in conversation. While at home he frequently expressed a desire to go home. It was thought he had auditory and visual hallucinations. He complained that some one was trying to get him into trouble. On admission to this hospital he was quiet and affable, but showed a marked deterioration, especially in the memory field. He was slightly confused and markedly disoriented. Attention was extremely difficult to gain and hold; he showed considerable paresis of thought with a tendency to repetition of questions, and words, with more or less incoherence. For the greater part of the time he remained quietly in bed, giving little trouble except for occasional untidy habits. He had no insight into his condition; judgment was markedly impaired, but no definite delusions were elicited. He had some suspicious ideas, but these were not clearly expressed or fully formulated.

Examination.—He was fairly well developed and moderately nourished. On the left side was an inguinal hernia. Supraclavicular and infraclavicular fossae were prominent on both sides. Heart: the apex was palpable in the fifth left interspace. There were no abnormal heart sounds. The peripheral arteries showed marked sclerosis. The blood pressure was: systolic, 210; diastolic, 98. The abdominal organs were normal. Urine: it contained albumin and a few hyaline casts. The Wassermann reaction in the blood serum was negative. There was slight ptosis; arcus senilis was present. The pupils were regular and reacted to light and accommodation. He could not hear a watch tick when held close to his ear. Sense of taste and of smell were normal. There were no disturbances of motility and sensibility. The knee jerks were slightly dimin-

11. Ernst: Missbildungen des Nervensystems, in Schwalbe: Morphologie der Missbildungen des Menschen und der Tiere, Lief., Part 3, No. 2, 1910.

12. Schwalbe und Josephy: Die Missbildungen des Kopfes, in Schwalbe: Morphologie der Missbildungen des Menschen und der Tiere, Part 3, No. 2, 1913.

ished. He showed a slight speech defect and inequality of the pupils but no definite paralytic symptoms in the arms or legs. He frequently stated that his head seemed twitchy. As far as surroundings were concerned he was disoriented, and only approximately correct in the matter of time.

Course of Disease.—July 9, 1920: The patient had a typical grand mal convulsion while in the dining room. Sept. 22, 1920: During September the patient

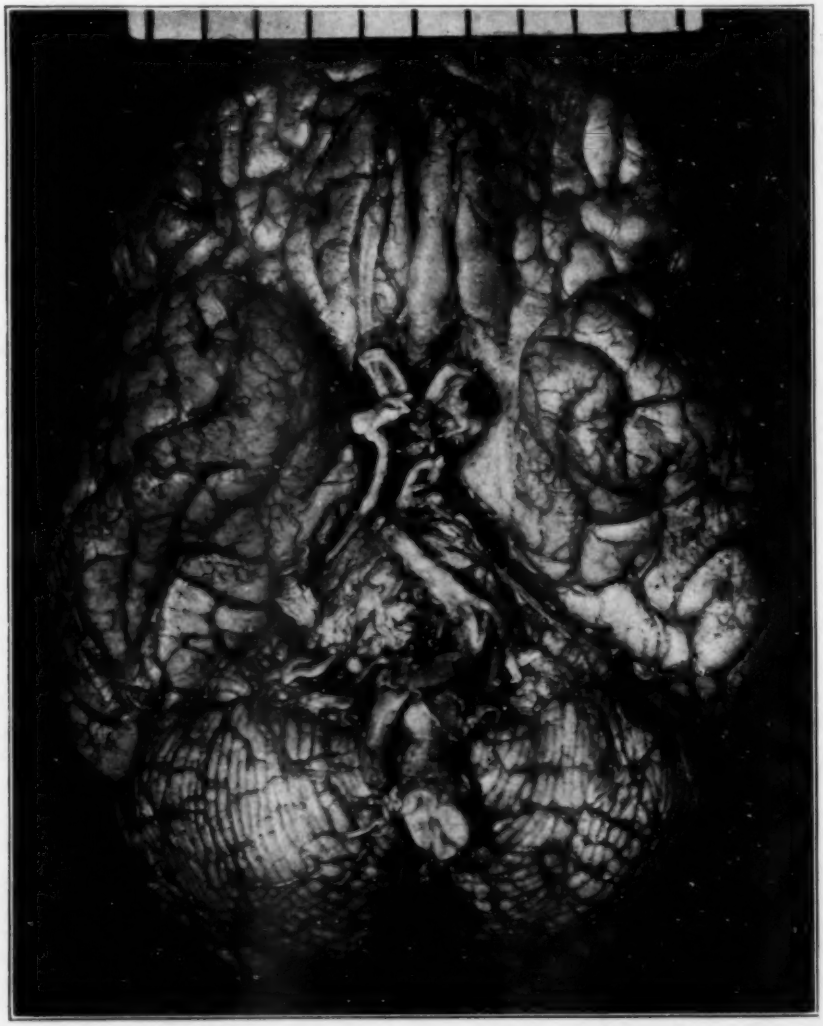

Fig. 2.—Softening of the left olfactory lobe.

had several convulsions, followed by much confusion. Nov. 3, 1920: The patient died at 3 a. m., the cause of death being given as chronic diffuse nephritis and arteriosclerosis.

Postmortem Examination.—Necropsy was performed five hours after death. Important anatomic diagnoses were: Decubitus in trochanter and sacral regions,

chronic adhesive pleuritis, old tuberculous process of the right apex, early bronchopneumonia, sclerosis of aorta and coronary arteries, chronic diffuse nephritis, fatty change of liver, chronic pachymeningitis and leptomeningitis, sclerosis of cerebral arteries, left olfactory bulb missing and softening of the left olfactory tract.

Head and Brain: The calvarium was dense; diploë was absent. The dura mater was thickened and adherent to the calvarium throughout. Pachyonian granulations were prominent. The brain weighed 1,410 gm. The pia mater was slightly thickened and opaque along the course of the large vessels. The vertebral arteries were markedly sclerotic on both sides, and noticeably dilated at a point 2 cm. below the junction. The basilar artery presented considerable sclerosis with whitish-yellow patches, which were for the most part calcified. The carotids and major arteries of the cerebrum and cerebellum were all thickened, atheromatous and calcified patches being strewn throughout. The right posterior cerebral artery was given off from the carotid, and a posterior communicating artery was found between the posterior cerebral artery and the terminal branching of the basilar artery.

The left olfactory bulb presented well marked degeneration. The bulb was replaced by a thin grayish thread, embedded in a thickened pia mater. The left olfactory tract was thinner than the right, grayish-yellow and extremely soft in consistency. The lateral olfactory striations could be traced for a distance across the substantia perforata anterior, toward the lobus pyriformis.

The convolutions did not appear to be atrophic, except in some small regionary areas. The lower surface of the middle frontal convolution on the left side was atrophic and brownish, suggesting a softening. On the lateral surface of the left occipital lobe was also found a small softening. The convolutional pattern was not much different on either side. With the exception of the anomaly of the circle of Willis, described in the foregoing, there were no noticeable malformations in the central nervous system. The lobus pyriformis, gyrus hippocampi, cornu ammonis, and gyrus fornicatus presented nothing unusual. The pons, cerebellum, medulla and spinal cord presented nothing remarkable.

Microscopic Examination.—The limbic lobe and the other important cortical areas were studied by the same methods as in Case 1. The arteries of the pia mater and cortex showed sclerotic changes. Proliferation of adventitia, proliferative as well as regressive alterations of the endothelium, occasional calcification of vessel walls and arteriofibrosis were encountered throughout. Occasionally small cortical vessels presented the appearance of hyaline degeneration. Irregular thickening of the marginal neuroglia network, a tendency toward wedgeshaped marginal gliosis, perivascular, cellular and fibrillar gliosis were also common findings. Focal areas of softening were observed in the left frontal pole and left occipital lobe. Perivascular thinning of the myelin sheaths was noted throughout, particularly in the left frontal lobe. In the neighborhood of the sclerotic vessels the nerve cells displayed various stages of regressive changes. A majority of the cells were shrunken with eccentric, irregular, chromophilous nuclei. Fatty pigmented degeneration was found in conjunction with the sclerotic cell changes. In short, changes encountered in general indicated an arteriosclerotic nature of the alteration.

Section of the olfactory tract of the left side revealed a well marked degeneration of the myelin sheaths, as well as of the axis cylinders. Few fibers remained uninvolved at the level of the olfactory trigon.

In the lobus pyriformis of the left side a remarkable alteration was encountered. The plexiform layer had become considerably narrower. The cells of the clusters were noticeably decreased in number, some clusters having apparently disappeared; some cells were shrunken with dark eccentric nuclei, while others were slightly swollen with chromatolysis and occasional vacuole formation. The equal sized pyramidal cells were greatly reduced in number, those remaining presenting various grades of degeneration; also slightly swollen cell bodies with chromatolysis and eccentric nuclei were encountered. The cells of the fusiform layer were fairly well preserved, although a number of cells showed marked regressive changes.

The tangential fibers were greatly involved; those remaining ran irregularly, and some were markedly thickened. In the layer of cell clusters and the lamina of pyramidal cells Weigert preparation showed marked reduction of radial, as well as of inter-radial, fibers. Quite large sized blank areas, without demonstrable fibers, also came to view. The nerve fibers in the medulla were apparently fewer than in the corresponding part of the other hemisphere.

The neuroglia fibers were increased, not only in the cell-free border, but also in the deeper cortical layers. The fibers were thicker than those observed in Case 1, and the network less dense. A considerable number of satellite cells were encountered, especially around the nerve cells of the deeper layers. Some cells were accompanied by more than nine satellites; two or three satellites were occasionally seen along the apical prolongations. The glial reaction, therefore, indicated a relatively fresh and still progressive degenerating process.

The blood vessels were more or less sclerotic, showing both regressive and progressive changes. However, the cell and fiber degeneration did not seem to be associated with the vascular alteration. The parenchymatous devastation was diffuse throughout the anterior part of the lobus pyriformis. The alteration of this particular part, described in the foregoing, should be considered as a secondary degeneration following the unilateral softening of the olfactory bulb and tract.

The cornu ammonis and the rest of the limbic lobe showed a slight degeneration, both sides being equal in intensity. The cell and fiber degeneration was usually found around, or in the neighborhood of, the sclerotic vessels. Most of the cells showed cell sclerosis combined with fatty pigmented degeneration, while no swollen cells with vacuoles or chromatolysis were encountered. There was slight fibrillar and cellular increase of neuroglia, especially along the cortical margin and around the sclerotic vessels. These changes were, in all probability, due to the arteriosclerotic condition of the vessels. It was almost impossible, however, to determine whether or not the olfactory degeneration displayed any secondary changes in this part of the cortex.

COMMENT ON CASE 2

Undoubtedly Case 2 was one of arteriosclerotic brain disease. The lesion of the left olfactory lobe should, by all means, be considered as softening due to arteriosclerosis. The whole picture of the lesion found in the homolateral lobus pyriformis suggested secondary degeneration following the primary involvement of the olfactory lobe. The history of the case revealed nothing abnormal concerning the olfactory sense of the patient. It is doubtful, however, whether the sense of smell was tested unilaterally.

The patient in Case 2 had typical epileptiform convulsions. The patient in Case 1 and Tanaka's patient were epileptic subjects. It is a question whether or not the secondary involvement of the cortical olfactory center might cause the epileptiform seizures. Both Tanaka's case and Case 1 were of a congenital nature and, therefore, the seizures might be attributed to it, as defective subjects are liable to show epileptic convulsions. The epileptic seizures of the patient in Case 2 might also be attributed to the arteriosclerotic condition, as this is often the case. To answer this question a study of cases of uncomplicated olfactory degeneration is desirable. For this purpose I searched 2,230 necropsy reports of the Danvers State Hospital Laboratory material, and found a case showing bilateral olfactory degeneration (of unknown nature) without arteriosclerosis or recognizable malformations. This patient (No. 18596, necropsy No. 1824) showed an absence of the olfactory sense, and the most typical acquired form of epileptic convulsions. The brain, however, was in such a condition that careful study of the finer structure was impossible, and nothing definite could be ascertained. However, I am inclined to believe in olfactory center degeneration as the possible cause of epilepsy, rather than as a coincidental complication.

SUMMARY

Although certain parts of Broca's limbic lobe are known to govern the sense of smell, opinions as to the exact area of the cortical olfactory center are manifold.

Gorschkow [13] demonstrated, by experiment, that the ablation of the lobus pyriformis would be attended by loss of the sense of smell on the same side and diminution on the opposite. In a case of tumor at the base of the right temporal lobe, destroying the uncus and gyrus hippocampi, reported by Siebert,[14] substances used to test the olfactory sense were falsely interpreted. In Jackson and Beevor's [15] case of tumor at the tip of the temporal lobe, involving the anterior end of the lobus pyriformis and the nucleus amygdalae, the patient was said to have been constantly afflicted with a sense of horrid smell. Cajal, from histologic study of human and vertebrate brains, reached the conclusion that the important olfactory center lies in the regio olfactiva of the lobus pyriformis, and that the cornu ammonis, focus spheno-occipitalis, the subiculum and the focus praesubicularis represent probably the

13. Gorschkow: Uber Geschmacks- und Geruchscentren in der Hirnrinde, Inaug. Dissert., St. Petersburg, 1901; abstr. Neurol. Centralbl. 1901.

14. Siebert: Ein Fall von Hirntumor mit Geruchstäuschungen, Monatschr. f. Psychiat. u. Neurol. **6**:1899.

15. Jackson and Beevor: Case of Tumor of the Right Temporo-Sphenoidal Lobe Bearing on the Localization of the Sense of Smell and on the Interpretation of a Particular Variety of Epilepsy, Brain **12**:346, 1890.

tertiary olfactory center. Bechterew regarded the lobus pyriformis, by experimental study on dogs, as the perception center, and the neighboring regions, such as the subiculum and cornu ammonis, as the conception center. Campbell concluded that the lobus pyriformis must almost certainly be regarded as the chief olfactory center.

Hill,[16] on the other hand, pointed out that in some anosmatic animals the lobus hippocampi attains a moderate degree of development, and therefore he believed that this lobe cannot exist for the control of this function solely. He attached far more importance to the fascia dentata of the cornu ammonis, because this is the only structure in this region which is wholly wanting in anosmatic animals. Edinger,[17] also from the comparative anatomic standpoint, seems to believe that the cornu ammonis is the chief cortical olfactory center.

In both cases of unilateral olfactory lobe involvement, herein presented, I found a well marked degeneration of the anterior part of the lobus pyriformis. The first case was, however, that of an epileptic subject, and therefore the changes might be of an epileptic nature and not of a secondary degeneration. The second case was undoubtedly that of an arteriosclerotic brain disease, but the changes in the lobus pyriformis were different than in other parts, indicating their secondary nature. Considering these two cases, it is not unreasonable to conclude that the degeneration of the lobus pyriformis in the first case was also secondary. From the study of these two cases, and also from data presented by various observers, I am inclined to believe that the lobus pyriformis must be the chief olfactory center in the human brain.

The cornu ammonis and the rest of the limbic lobe showed little evidence of degeneration, and I have nothing to offer, from the present study, as to the functional importance of these parts.

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16. Hill: The Hippocampus, Philadelphia Trans. **184**:1893.
17. Edinger: Vorlesungen uber den Bau der nervosen Zentralorgane, 1911.

A CASE OF FRIEDREICH'S ATAXIA

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AND
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Friedreich's ataxia is still a rarity, and the opportunity to examine the spinal cord in an early stage of the disease does not often present itself. The following case is of interest because it occurred in a boy, aged 11 years, who died of an acute intercurrent disease. He had a younger brother who also had this familial affection. The fact that they were negroes perhaps lends additional interest, for Friedreich's ataxia is probably even more rare in the negro than in the white race.

The problem which the disease presents is not so much the distribution of the lesions as the causation and exact nature of the affection. As is well known, the disease process is not confined to the posterior columns; it is more diffused, and is evidently a degeneration with neurogliar proliferation, but the causation is obscure and has given rise to a variety of theories, none of which is particularly convincing. The fact that it occurs in several children of the same family, suggests, of course, some obscure familial defect; but the additional fact that numerous brothers and sisters often escape, indicates that the cause, whatever it is, is strangely limited or selective in its action. Gowers' theory that the disease is due to an "abiotrophy," or developmental defect, has been received with some favor, although it is not so much an explanation as a mere statement of a fact, for the problem remains as to what is meant by an "abiotrophy," and why it acts, as in the present instance, in only two out of five children; and in still another instance, which one of us has recently seen, in two out of eight children. That the disease is not due to an infection seems certain. Syphilis can be ruled out.

There are some physiologic problems also, as, for instance, the cause of the ataxia, the possible rôle of the cerebellum, and the absence in many cases of pronounced involvement of sensation. Possibly the degeneration so frequently found in the cells of Clark's column and in the direct cerebellar tract may explain the peculiar character of the ataxia. That it is not due to cerebellar disease is indicated by the fact that the cerebellum has not been found diseased in most of the cases examined. The absence of tactile and other modes of anesthesia has been ascribed by A. W. Campbell [1] to the escape of the posterior roots and the sensory tracts in the cord. The spinothalamic tract is not

1. Campbell, A. W.: M. J. Australia **1**:135 (Feb. 17) 1917.

involved. The degeneration sometimes seen in the posterior roots and even in the cells of the posterior ganglions may be merely a consequence of the posterior column lesions. A possible resemblance of the lesions to those of multiple sclerosis has been suggested. The two lesions have, however, little in common. The lesions of multiple sclerosis are primarily focal in character and irregular in distribution. Friedreich's ataxia is a system disease, but the widespread glial activity shown in the present case at least suggests that there is also here, in some sense at least, a primary gliosis.

The location of the lesions in the posterior columns in the present case suggests a resemblance to what is seen in locomotor ataxia, but that the disease is not tabes is generally recognized. We do not mean to stress this resemblance here, but we venture to call attention to what seems to be an involvement especially of the third series of fibers as observed in the process of myelinization. This resemblance is shown on comparing the photographs with those of Trepinski, a student of Flechsug.[2] Whether it has any significance, we do not know.

The involvement of one of these systems, usually the third, to the exclusion of the remainder of the posterior column fibers, is common in tabes, but probably is not infrequent in other forms of posterior column gliosis. In this case it may mean nothing more than a fiber destruction corresponding to the posterior root involvement. Which is the earlier of the two is a subject for speculation. It is interesting to note that the microscopic structure of the replacement glia is here totally different from that occurring in tabes.

Much has been written about the possible relationship of Friedreich's ataxia to Marie's so-called cerebellar ataxia. Such a relationship is not indicated in the present case, for neither the cerebellum nor the inferior cerebellar peduncle is involved. If there is any relationship between the two diseases it would seem to depend on some common obscure developmental defect acting on different structures in different cases.

It has been suggested [3] that the location of the lesions in the posterolateral regions of the cord may be due to a less adequate blood supply, the supposition being that therefore the resistive power is less. This idea was suggested by Williamson, who pointed out that these regions receive their blood supply from small meningeal branches, instead of from a deep central artery as in the anterior and central parts of the cord. But this is not an adequate anatomic reason for supposing differences in the extent of vascularization of the parts of the cord, unless such a conclusion is to be drawn from the situation of the cord lesions in

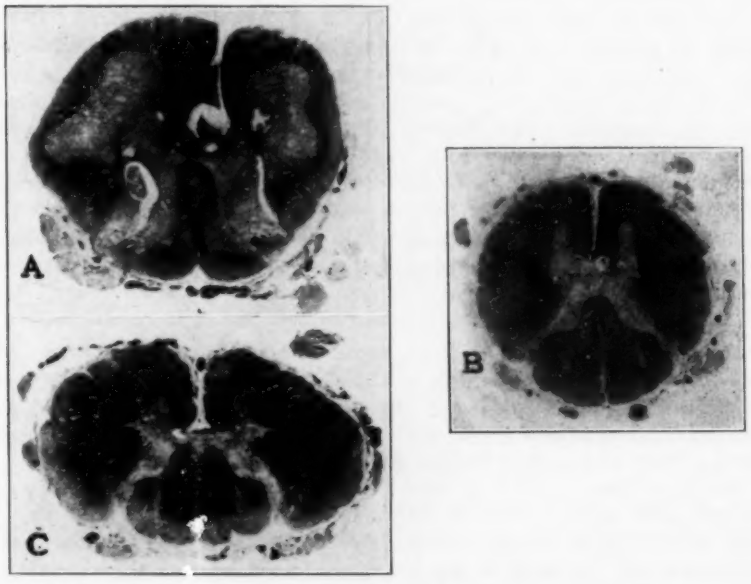
2. Barker: The Nervous System, New York, G. E. Stechert & Co., p. 430.

3. Williamson: Diseases of the Spinal Cord, New York, Oxford University Press.

pernicious anemia, as Campbell has suggested. The lesions in Friedreich's ataxia are, however, quite unlike those in pernicious anemia.

CLINICAL REPORT BY DR. LLOYD [4]

Family History.—The two brothers, one of whose cases is here reported, were full-blooded negroes. They were well developed and intelligent for their age. The father, aged 39, and the mother, aged 35, were in good health. The blood of both the mother and an older daughter gave a negative reaction to the Wassermann test. The blood of the father could not be secured, but the test of the mother's blood was more important for determining the question of congenital syphilis. The blood and spinal fluid of both the patients were negative. The mother had had five children, a daughter, aged 14, living and



Weigert sections of different levels of the cord, showing involvement of the third myelogenetic system in the posterior columns. The peripheral paleness is an artefact.

well; two sons, the patients, and another son who had had convulsions and died at 3 years of age; and a daughter, aged 3, who was living and well. There had been one miscarriage. Otherwise the family history was unimportant. There had been no ataxia in collateral branches, so far as could be ascertained.

Case 1.—J. C., the elder brother, aged 11 years, was the one whose brain and cord were examined. He was born in normal labor, and walked and talked

4. From the Neurological Wards of the Philadelphia General Hospital. The patients were shown at a meeting of the Philadelphia Neurological Society, Nov. 21, 1919. See report of transactions, Arch. Neurol. & Psychiat., March, 1920.

at the usual age. He had convulsions in infancy and the usual diseases of childhood. He went to school for a while and did well. The ataxia began when the boy was 8. He became awkward in the movements of his legs, falling on his knees when attempting to walk. Scoliosis was present. When he was admitted to the hospital, in the autumn of 1919, his gait was very ataxic, he kept his feet far apart, swayed violently, and required assistance in walking. It was not the gait of locomotor ataxia. He had the Romberg sign, and well marked adiadokokinesis. The finger-to-nose and heel-to-knee tests showed ataxia. The knee and Achilles' reflexes were absent, and there was no Babinski reflex. The abdominal and cremasteric reflexes were preserved. Superficial sensation in all its modes was normal, that is, to touch, pain, heat and cold. There was apparent loss of ability to recognize a tuning fork held on the radius, ulna, or tibia. There was no shortening of the plantar arch, no hammer toe, as reported in some cases; no muscular atrophy, no pain, no girdle sense, no loss of spincter control. Speech was hesitating and staccato rather than drawling. Vision was normal, the pupils equal, the reaction to light sluggish, the action on accommodation and convergence prompt. Dr. Shannon reported a curious lack of pigmentation in the upper nasal quadrant of the iris of the right eye, also in the upper nasal and lower temporal quadrants of the left iris, suggesting iritic atrophy. On distant fixation the left eye rotated outward, although on close fixation fair convergence was obtained, indicating a condition of exophoria. This measured about 12 degrees with the Maddox rod. Ataxic nystagmus was not elicited. In the right eye the media were clear, the disk discolored and somewhat atrophic, the margins clearly outlined, the vessels moderately contracted, the macula normal; there were no changes in the periphery of the fundus. The left eye was in a similar condition. A few months after his admission to the hospital this boy died of influenzal pneumonia.

Case 2.—The younger brother, aged 9, was born in normal labor, walked and talked at the usual age, went to school for a while and was considered bright. He had convulsions at the age of 2 years and the usual diseases of childhood. The ataxia began when he was 6 years old and when he was recovering from an attack of typhoid fever. His gait and most other symptoms were similar to those of his brother. He had the ataxic gait, with swaying movements and unsteady station, which, however, was not increased on closing his eyes. The deep reflexes were abolished. There was no pain and no loss of superficial sensation in any of its modes; there was slight loss to the tuning fork. Unlike his brother, he did not have scoliosis. There was no muscular atrophy, no loss of sphincter control. Speech was slightly hesitating. The eyes were in a similar condition to that of his brother. Nystagmus was seen when the eyes were fixed on a moving object, especially in extremes of rotation. The light reflex was sluggish, accommodation normal.

COMMENT

It is noteworthy that speech defects and nystagmus were not marked in these boys, but these symptoms are sometimes rather late in appearing in Friedreich's ataxia. The slight appearance of optic atrophy is also noteworthy. Several cases have been reported in which the light reflex was lost, and Gowers thought that this was an evidence of syphilis. It might occur in juvenile tabes, but the present cases were evidently not instances of that disease.

The occurrence of convulsions in three children in this family is not to be overlooked. The two boys and another brother had been thus affected. It may at least indicate an unstable nervous system.

HISTOLOGIC REPORT BY DR. NEWCOMER [5]

The gross appearance of the brain and cord was not remarkable. Sections were cut from the various cord levels, from the cerebellar cortex, including the anterior and posterior superior worm, from the cerebellar nuclei and from six areas of the cerebral cortex. In sections from the cortex stained with toluidin blue, there were some distinct vessel fibrosis and perivascular infiltration, partly with leukocytes and partly with round cells which were perhaps glial in origin. There was a slight general gliosis, some areas contained nerve cells with a number of satellite cells, rarely more than six or seven, but enough to attract attention. The nerve cells and sections from the basal ganglions showed nothing remarkable, except that not infrequently they were almost filled with pigment. The sections from the cerebellum were normal. In the superior worm, the Purkinje cells were normal and as numerous as usual. In the medulla, the nerve cells contained considerable pigment, less in the olive. Sections from the cord showed marked pigmentation of the anterior horn cells. The lateral horn cells showed some simple chromatolysis. There were no normal Clark's column cells in numerous sections from the dorsal cord. They were either absent, or, when present, were much diminished in number and markedly sclerosed; many gave evidence of considerable chromatolysis. There was considerable gliosis of the column. In the cord there was the same vessel fibrosis, but not the infiltration seen in the cortex.

Sections stained with phosphotungstic acid hematoxylin, with the exception of the cord, showed nothing further that was remarkable. Sections of the cord gave evidence of a considerable gliosis of the posterior columns which involved, in all the levels, substantially the region of the third myelogenic system as described by Trepinski,[2] and in which the nerve fibers were practically all gone. This distribution is seen in the photographs of the Weigert specimens. These illustrations show the posterior column degeneration only. In the rest of the posterior columns there was a gliosis of less intensity. The gliosis in the region of the greatest scarring consisted of a dense fibrous mat with rather small inactive glia cell nuclei—a mat of whorls and irregularly arranged glia fibers. There were some spider cells, but none of the columnar fiber arrangement so typical of tabes. In the less involved regions of the posterior columns the gliosis was

5. From the Laboratory of the Pennsylvania Hospital, Department for Mental and Nervous Diseases, Philadelphia.

also largely of fibrous character, but spider cells were more numerous. In all the levels of the cord there was some gliosis of the crossed pyramidal tract. This gliosis was fibrous in character but contained a number of rather large spider cells. Nerve fiber replacement was appreciable in the center of the tract. At the border of the cord in the region of the dorsal cerebellar tract there was a certain amount of spider cell gliosis, and the glia fiber strands were heavier and more conspicuous than anywhere else along the border of the cord. In the region of the direct pyramidal tract there were some gliosis and large spider cells. This slight gliosis of the direct pyramidal tract decreased as we progressed toward the dorsal and lumbar region. There was more gliosis than usual in the region of the anterior commissure. In the gray matter of the cord there was strikingly more than the usual number of glia cells—many of them were large spider cells quite active in appearance. The gliosis in the gray matter was greatest, both fibrous and cellular, in the lower region of the cord, dorsal and lumbar. It was less in the posterior horns than elsewhere. There was considerable fiber loss with extensive spindle cell replacement in the posterior nerve roots and considerable glia fiber invasion of the proximal portion of the nerve roots. The fiber loss was greatest in the cervical region, involving most of the fibers and decreased gradually to become least in the sacral region where only a few fibers were gone. The anterior roots were intact.

With the acid fuchsin—light green stain, the extensive cord gliosis was distinct, and it was possible to estimate the fiber loss in the direct cerebellar tracts as being about one sixth. The crossed pyramidal tracts were about the same. The posterior column gliosis was, if anything else, less than this except in the region of the third system in which the fiber loss was practically complete. In the ventral field more than half the fibers were gone.

There was thus a general vessel fibrosis and some perivascular infiltration, of which at least the latter may be considered as due to the terminal disease (influenzal pneumonia). There was a general marked nerve cell pigmentation and a gliosis of the gray matter of the cortex and cord, and in addition there was a tract gliosis involving almost completely the fibers in the posterior columns having the position of the third myelogenic system and slightly the direct cerebellar and crossed pyramidal tracts. There was extensive posterior nerve root degeneration. The involvement of the direct cerebellar tract was too slight to show in the Weigert preparations of the restiform body, and there was nothing abnormal in the appearance of the dorsal portion of the superior worm, nor was there any gliosis of this or other portions of the cerebellar cortex.

COMPARATIVE RESULTS OF COLLOIDAL MASTIC AND COLLOIDAL GOLD TESTS*

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Although the colloidal mastic test, first introduced by Emmanuel,[1] and later modified by Cutting,[2] has been in limited use for several years, the question of its value as compared with that of the colloidal gold test is still an unsettled one. Considering the more complicated and uncertain methods of preparing the reagents used in the colloidal gold test, the simplicity and uniformity of the mastic reaction has much to recommend it. If it can be shown that the information provided by the mastic test is equivalent to that given by the gold test, or that the mastic test may in some cases provide information which the gold test fails to give, the test deserves wider use than is now made of it. In either case, the mastic test should prove of value not only when facilities for performing the gold test are lacking, but also as a check on the more complicated gold reaction in the laboratories where this test is now employed.

Both the gold and mastic reactions apparently depend on colloidal phenomena. The gold solution undergoes quantitative colorimetric changes leading up to complete precipitation, while the suspension of finely divided colloidal mastic particles undergoes quantitative flocculation, also culminating in complete precipitation. Resins other than mastic may be employed for the preparation of the colloidal suspension, the successful substitution depending on a proper adjustment of other reagents employed in the test, to balance variations in the amount of free acids present in other resins. It seems probable that these free acids exercise a dominating influence on the reaction.

Recent authors have thoroughly reviewed the literature, making a further complete survey unnecessary. Stanton[3] and Camp[4] conclude that the results obtained with the mastic test are in close agreement with those obtained with the gold test.

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* From the Syphilis Department of the Medical Clinic, Johns Hopkins Hospital.

1. Emmanuel, G.: Eine neue Reaktion zur Untersuchungen des Liquor cerebrospinalis, Berl, klin. Wchnschr. **52**:792, 1915.

2. Cutting, J. A.: A New Mastic Test for the Spinal Fluid, J. A. M. A. **68**:1810 (June 16) 1917.

3. Stanton, J. M.: Concerning the Colloidal Mastic Test, Arch. Neurol. & Psychiat. **4**:301 (Sept.) 1920.

4. Camp, C.: Colloidal Mastic Test on Cerebrospinal Fluid, Am. J. Syph. **4**:301, 1920.

In order to secure further information regarding this still unsettled point, we have studied the spinal fluids of 311 patients. Gold [5] and mastic tests were compared with each other and with the cell counts, globulin reactions and Wassermann tests in all the fluids. The material is almost entirely from the syphilis department, and consists of fluids from patients exhibiting various forms of syphilis. A small number of fluids from nonsyphilitic patients, on whom puncture was performed to rule out neurosyphilis, has also been examined. Of the 311 fluids, there was complete agreement between the gold and mastic tests 234 times. In the remaining seventy-seven instances in which there was disagreement, the discrepancies have been analyzed.

TECHNIC

The technic which we have employed in the mastic test is that described by Stanton.[3] It is essentially as follows: Ten grams of commercial gum mastic are dissolved in 100 c.c. of absolute alcohol, and the resultant cloudy fluid filtered several times until a clear straw colored solution is obtained. This stock solution is kept in glass stoppered bottles at room temperature. The emulsion is prepared with 1 c.c. of the stock solution added to 9 c.c. of absolute alcohol, which is then added, with gentle mixing, to 40 c.c. of once distilled water.

In setting up the test ten small tubes are employed. To the first tube in the series are added 1.5 c.c. of a stock salt solution (99 c.c. of 1.25 per cent. sodium chlorid solution plus 1 c.c. of 0.5 per cent. potassium carbonate solution), and to each of the remaining tubes 1 c.c. is added. In the first tube there is placed 0.5 c.c. of the spinal fluid to be tested. After mixing this dilution of spinal fluid, a titration is made throughout the series by transferring 1 c.c. from the first to the second tube, from the second to the third, etc., 1 c.c. being finally discarded from the last tube. Finally, 1 c.c. of the mastic emulsion is added to each tube. After mixing, the tubes are allowed to stand over night at room temperature, and the results are read in the morning. No special precautions regarding absolute cleanliness of glassware, such as are necessary for the gold test, need be observed.

In recording results, previous experimenters have used either the terms positive and negative, or, as later suggested by Smith,[6] a numerical scale denoting degrees of flocculation and precipitation, as follows: No change is indicated by 0; loss of opalescence and very slight precipitation by 1; milky fluid with distinct precipitation by 2; marked precipitation with turbid fluid by 3; and complete precipitation by 4.

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5. The colloidal gold tests were all performed in the laboratory of the Phipps Psychiatric Clinic of this hospital, under the supervision of Dr. Phyllis Greenacre, for whose cooperation we are deeply grateful. The technic employed is that described by L. D. Felton in "A Study of the Specificity of the Colloidal Gold Reaction from the Physicochemical Standpoint," Trans. Sect. Path. and Physiol., A. M. A., 1917, p. 73.

6. Smith, E. R.: The Mastic Reaction on the Cerebrospinal Fluid, Med. Rec. **92**:675, 1917.

With this scale it is possible to chart curves which correspond in type to those of the gold test, but as they differ numerically, 4 denoting in the mastic test complete precipitation, which in the gold test is recorded as 5, we deemed it expedient as a preliminary to our work to construct a scale which would exactly correspond to that employed everywhere in the gold test. By observing the reactions obtained in a number of fluids in parallel series of gold and mastic tests the following scale was arrived at: 0, opalescence, no change; 1, milky fluid with no precipitation; 2, milky fluid with slight precipitation; 3, milky fluid with moderate precipitation; 4, cloudy fluid with almost complete precipitation; and 5, clear fluid with complete precipitation. With this scale mastic reactions recorded as 2 may occur in normal fluids with about the same frequency as in the gold test, and such reactions are therefore regarded as within normal limits. Fluids which produce moderate precipitation, however, as denoted by a reading 3, we have tentatively regarded as abnormal for reasons which will be given in the analysis of this type of reaction. Slight color changes occurring in normal fluids, as observed in the gold test, occur more frequently with some preparations of colloidal gold chlorid solutions than with others, but a color change reading as high as 3 is regarded as abnormal and is recorded as a syphilitic zone curve.

We have observed three types of curves with the mastic test: a paretic curve, a negative curve and a "mastic 3" curve. In only one instance was a curve of the gold syphilitic zone type observed. For the purpose of comparison with the gold reaction, we have arranged our results in seven groups as follows:

RESULTS OF AUTHORS' TESTS

Gold and Mastic Curves Both Negative.—In this group are 203 of the fluids examined. In general, there was complete agreement with the clinical findings and with the other tests of the cerebrospinal fluid. In only one instance was there a positive Wassermann reaction in the fluid with both colloidal tests negative; this was a fluid with 24 cells and a positive globulin reaction in a patient with secondary syphilis with irregular pupils and sluggish reflexes. Cell counts were not made in all fluids, but in thirteen instances there was an increased cell count or an increased globulin content or both. The diagnoses of the patients in this group were: primary syphilis, 11; secondary syphilis, 45; tertiary syphilis, 42; syphilis Wassermann, 34; latent syphilis, 7; congenital syphilis, 6; neurosyphilis (mostly treated cases), 21; undiagnosed (except in one instance all referred from other clinics for spinal puncture), 14; and nonsyphilitic patients, 23. The nonsyphilitic patients composed a diagnostic group including normal persons investi-

gated for familial syphilis, neurasthenia, lethargic encephalitis, transverse myelitis and other neurologic affections. Practically all the syphilitic patients had received some treatment before the time of puncture, which probably accounts for some of the negative curves in those fluids showing other minor abnormalities.

TABLE 1.—Gold and Mastic Curves Both Paretic *

Case No.	Diagnosis on Admission. Form of Syphilis	Blood Wassermann Reaction	Cerebrospinal Fluid						
			Cells	Globulin	Wassermann 0.2 C.c.	Wassermann 0.4 C.c.	Wassermann 1.0 C.c.	Gold Curve	Mastic Curve
B3	Central nervous system, unclassed	3	N. C.	++	4	4	4	5554410000	5554310000
Ax21	Wassermann	4	N. C.	++	4	4	4	5555555221	5532100000
Ax24	Tabes	4	N. C.	++++	4	4	4	5555542100	5554321000
Bx7	Tabes	4	N. C.	++++	4	4	4	5555532100	5554321000
Cx5	Paresis	4	N. C.	++++	4	4	4	5555555821	5555554321
Cx24*	Central nervous system, unclassed	0	N. C.	++++	4	4	4	5554*310000	5554321000
Cx31	Paresis	4	N. C.	+++	4	4	4	5555521000	5555321000
Dx14	Paresis	4	N. C.	++++	4	4	4	5554100000	5555*2100
Dx19*	Secondary neurorecurrence	4	N. C.	+++	0	4	4	55442*21100	5553210000
Ex32	Wassermann	4	14	++	4	4	4	5442210000	5555432100
Fx13*	Central nervous system, treated	0	5	++	0	4	4	554*4*4*21000	5553210000
Fx15*	Paresis, treated	0	2	+	0	0	0	5555210000	5554321000
Kx13	Nonsyphilitic ?	0	N. C.	++	0	0	0	5555432200	4321000000
Z1	Paresis	4	N. C.	++++	4	4	4	5555431000	5555432000
Z3	Paresis	4	62	++++	4	4	4	5555432000	5555521000
Z4	Paresis	4	N. C.	+++	4	4	4	5555552100	5555321000
Z5*	Taboparesis	0	20	+++	4	4	4	5554422100	5555532100
Z6*	Wassermann	4	N. C.	++++	4	4	4	5555521000	5555321000
Hx18	Wassermann	4	14	++++	4	4	4	5542210000	5555321000
Hx23	Nonsyphilitic, lethargic encephalitis	0	7	++++	0	0	0	5433210000	554*3211000
Hx30	Central nervous system, unclassed	4	11	+++	0	3	4	5544320000	5554322100
Ix3	Central nervous system, meningovascular	2	26	++++	0	0	0	4442210000	5532200000
Ix5	Paresis	4	20	+++	4	4	4	5555531100	5555421000
Ix7	Central nervous system, vascular	4	90	+++	4	4	4	5444411000	4221000000
Ix10	Tabes	4	40	+++	4	4	4	5554411000	5532100000
Ix11	Tabes	0	20	+++	4	4	4	5543310000	5432100000
Ix25	Central nervous system, unclassed	4	0	+++	3	4	4	5555422100	4321000000
Jx1*	Secondary	0	98	++++	4	4	4	5555430000	5532100000
Jx6	Paresis	4	100	++++	4	4	4	5555554320	5555321000
Jx24*	Central nervous system, asymptomatic, treated	0	61	++++	4	4	4	4*4*44432*200	5555321000

* An asterisk after a case number signifies that the patient had been treated for syphilis by us.

* The globulin test was performed with Pandy's reagent. The Wassermann reactions were made with both plain alcoholic and 0.2 per cent. cholesterinized antigen.

* In this and the following tables, the diagnoses given are those made on the patient's admission, on the basis of clinical evidence. If neurologic symptoms or signs were absent, the diagnosis is allowed to remain unchanged, even when positive results in the spinal fluid examination show the presence of asymptomatic neurosyphilis.

Gold and Mastic Curves Both Paretic.—Of the thirty patients in the group (Table 1), only one was presumably nonsyphilitic; this was a case which had been diagnosed as lethargic encephalitis. In twenty-three of the twenty-nine syphilitic patients there was definite clinical and serologic evidence of neurosyphilis (paresis 9, tabes 4, unclassed neurosyphilis 10), while the remaining six were diagnosed as asymp-

tomatic neurosyphilis on the basis of blood tests and other spinal fluid abnormalities. A comparison of the gold and mastic curves in this group shows that they are almost parallel. The number of tubes in which complete precipitation occurred is sometimes greater with the gold, sometimes with the mastic test; but a fluid showing a very long or a very short paretic gold curve tends to show a mastic curve of the same general type.

Gold Curve Paretic, Mastic Curve Negative.—Only five patients fall into this group (Table 2). All of these patients were neurosyphilitic, and in all the clinical evidence, or that provided by other tests, showed that in these cases the gold curve is to be relied on rather than the mastic. Only one of the patients gave a positive Wassermann reaction of the spinal fluid, and at least two had normal cell counts.

TABLE 2.—GOLD CURVE PARETIC; MASTIC CURVE NEGATIVE

Case No.	Diagnosis on Admission. Form of Syphilis	Blood Wassermann Reaction	Cerebrospinal Fluid						
			Cells	Globulin	Wassermann 0.2 C.c.	Wassermann 0.4 C.c.	Wassermann 1.0 C.c.	Gold Curve	Mastic Curve
C14*	Tertiary	0	17	±	0	0	0	4553221000	2221000000
R4*	Wassermann	3	41	+++	4	4	4	5554210000	2111000000
S8*	Tertiary	4	N. C.	±	0	0	0	4442100000	2221100000
Ix24	Central nervous system, unclassed	4	0	+++	0	0	0	5554310000	2110000000
Ix29	Central nervous system, vascular	?	4	+	0	0	0	5443310000	2210000000

Gold Syphilitic Zone, Mastic Negative.—There were twelve patients in this group (Table 3), two of whom were definitely nonsyphilitic (one a normal person, one a case of multiple sclerosis). There was confirmatory evidence of damage to the central nervous system in the fluid Wassermann reaction of only two patients, although in three others the globulin test was strongly positive. One of the patients with a positive fluid Wassermann reaction showed a persistently positive blood Wassermann reaction as the only clinical suggestion of neurosyphilis. In the other there was no clinical suspicion of central nervous system invasion. An additional patient had neurosyphilis which had been consistently treated. It is possible that the syphilitic zone gold curve in some members of this group may be explained if the gold test was performed three or four days after the spinal puncture. The fluid is usually not collected in sterile tubes, and bacterial contamination may play some part in the production of gold curves of this type. The presence of the curve in a normal patient in whom syphilis can be excluded lends support to some such explanation.

Gold Syphilitic Zone, Mastic Paretic.—A comparison of this group with the last shows that in thirty-one instances in which a syphilitic zone gold curve was obtained, the mastic test was negative twelve times and paretic in type nineteen times. As stated in the foregoing, we have observed a syphilitic zone mastic curve only once. This was in a patient with cord tumor and xanthochromia. The spinal fluid showed no cells, a ++++ globulin reaction, a negative Wassermann test with all dilutions, a gold curve of the meningitic type reading 0001122411, and a mastic curve which read 224+5532100.

In fifteen of the nineteen patients in this group (Table 4) there was definite clinical evidence of neurosyphilis; and in three of the remaining four the other tests in the fluid substantiated the diagnosis of neurosyphilis. The exception proved to be a case of lethargic

TABLE 3.—GOLD CURVE SYPHILITIC ZONE, MASTIC NEGATIVE

Case No.	Diagnosis on Admission. Form of Syphilis	Blood Wassermann Reaction	Cerebrospinal Fluid						
			Cells	Globulin	Wassermann 0.2 C.c.	Wassermann 0.4 C.c.	Wassermann 1.0 C.c.	Gold Curve	Mastic Curve
H6*	Wassermann	1	N. C.	++	0	0	0	2321000000	2210000000
W11*	Wassermann	2	13	±	1	2	3	1123000000	2211200000
Ax4*	Wassermann	0	N. C.	0	0	0	0	223+3+110000	2211100000
Bx15*	Secondary	4	N. C.	+	1	4	4	1123110000	2222100000
Dx4*	Wassermann	0	N. C.	±	0	0	0	2355110000	2100000000
Dx5*	Secondary	0	N. C.	+++	0	0	0	2233100000	2220000000
Dx16*	Wassermann	4	N. C.	++++	0	0	0	2343100000	2211000000
Ex19*	Central nervous system	0	0	+	0	0	0	1233332000	2210000000
Ix1*	Wassermann	0	2	+	0	0	0	1233332000	1000000000
Ix6	Nonsyphilitic, normal	0	0	0	0	0	0	2332200000	1000000000
Ax17*	Secondary	0	N. C.	++	0	0	0	2445522100	2111000000
Jx23	Nonsyphilitic, multiple sclerosis	0	2	+	0	0	0	12332+00000	2210000000

encephalitis. In general, the paretic mastic curves obtained here were of a shorter type than those in the group in which the gold curve was also paretic.

These results permit the conclusions that a paretic type of mastic curve is even less significant of paretic neurosyphilis than a paretic gold curve; that it is commonly obtained in other types of neurosyphilis; and that it may be found, as may a similar gold curve, in nonsyphilitic neurologic diseases. In only two patients in this group was even a preliminary diagnosis of paresis permissible. However, in our opinion, it must be accepted that a paretic mastic test obtained in a syphilitic patient is indicative of definite damage to the central nervous system.

Gold Curve Negative, Mastic Paretic.—The fact that seventeen of the total number of patients studied have shown colloidal tests of this type lends support to the argument that the mastic test should occupy a more prominent place in the routine examination of the

cerebrospinal fluid. Inspection of the data regarding these seventeen patients (Table 5) shows that in eleven there was clinical evidence sufficient to support a diagnosis of neurosyphilis; in eleven the Wassermann test of the spinal fluid was also positive. Only three patients showed neither definite clinical nor other serologic evidence of central nervous system invasion.

Mastic Curve "3", Showing Moderate Precipitation in the First Few Tubes; Gold Curve of Various Types.—This group, comprising twenty-four cases, is of especial interest because of the borderline type of the mastic curve (Table 6). They were divided clinically as follows: primary syphilis, 1; secondary, 5; tertiary, 5; latent, 1; Wassermann positive, 7; cerebrospinal syphilis, 4; and nonsyphilitic, 1. Eighteen had received antisyphilitic treatment prior to the examination of the fluid. In only three instances was the Wassermann test of the fluid positive in any dilution. In one of these (P3) the gold curve was paretic in type; in another case (GX1) it was negative, although the globulin reaction was +++; and in a third (GX22) it was of the syphilitic zone type. In one case (AX7) the gold test gave a high syphilitic zone curve; the clinical diagnosis was neurosyphilis unclassed. In a case (CX18) with vascular neurosyphilis, the gold curve was also of the syphilitic zone type. One patient (DX9) was nonsyphilitic and had lethargic encephalitis; and seven showed no clinical or other

TABLE 4.—GOLD CURVE SYPHILITIC ZONE, MASTIC CURVE PARETIC

Case No.	Diagnosis on Admission. Form of Syphilis	Blood Wassermann Reaction	Cerebrospinal Fluid						
			Cells	Globulin	Wassermann 0.2 C.c.	Wassermann 0.4 C.c.	Wassermann 1.0 C.c.	Gold Curve	Mastic Curve
C4*	Wassermann	0	8	+	0	0	0	0113110000	4322111000
Ax26*	Central nervous system, meningitis	0	N. C.	++	0	0	0	2223222100	5543100000
Bx29*	Secondary	0	N. C.	++	0	0	1	1112333000	5432110000
Cx8*	Central nervous system, meningeal	0	12	++	0	3	4	2222411000	5310000000
Cx17	Paresis	4	N. C.	++++	4	4	4	1123221000	5554321000
Cx22*	Central nervous system, meningeal	0	N. C.	++	0	0	0	3345210000	5543210000
Ex14*	Wassermann	4	943	++++	4	4	4	0123330000	5555322100
Ex20	Unclassed (encephalitis ?)	0	2	+++	0	0	0	2244441110	5543211000
Ex28*	Wassermann	0	N. C.	+	1	3	4	1232221000	4321000000
Fx10*	Secondary	0	8	++	0	1	4	2+3+32+110000	5543200000
Fx23	Central nervous system, unclassed	0	8	++++	4	4	4	2223341000	5554321000
Gx6	Central nervous system, unclassed	4	0	+++	0	2	4	1223321000	4322100000
Gx11	Tertiary	4	26	+++	0	0	0	1133211000	4432210000
Gx23*	Tabes	0	5	++++	0	0	4	2244210000	5543210000
Gx28	Central nervous system, 8th nerve	0	6	++++	2	3	4	2332100000	5542210000
Gx31*	Wassermann	4	102	++++	4	4	4	1123210000	554+2100000
Hx12	Paresis	4	73	++++	4	4	4	2441111000	4455553210
Hx29*	Central nervous system, unclassed	0	35	++++	0	2	4	3333421000	5552221000
Z2	Tabes	4	N. C.	++++	2	4	4	2+443100000	5554321000

serologic evidence of neurosyphilis, rendering the value of the mastic test doubtful in those cases. In the remaining eleven patients, however, there were either clinical signs strongly suggesting neurosyphilis, or a definitely abnormal cell count and globulin content, or both. To sum up: In seventeen of the twenty-four cases in this group, other evidence of central nervous system invasion in addition to the borderline mastic test was present, while in seven cases such confirmatory evidence was absent. This leads to the conclusion that a mastic test of this type must be interpreted only in connection with the other clinical and serologic findings, but that in general it may be considered as a definite abnormality. It is of interest that in only four fluids showing

TABLE 5.—Gold Curve Negative, Mastic Curve Paretic

Case No.	Diagnosis on Admission. Form of Syphilis	Blood Wassermann Reaction	Cerebrospinal Fluid					Gold Curve	Mastic Curve
			Cells	Globulin	Wassermann 0.2 C.c.	Wassermann 0.4 C.c.	Wassermann 1.0 C.c.		
B19	Wassermann	4	7	±	4	4	4	1222100000	5543210000
M10	Central nervous system, meningeal	0	N. C.	++	0	2	4	1112100000	5543100000
Ax15*	Tabes	0	N. C.	+	4	4	4	0112221100	4553110000
Cx3	Congenital	4	N. C.	++++	4	4	4	0112211100	5553321000
Cx23*	Secondary	0	N. C.	±	0	0	0	0122100000	4322100000
Dx8*	Primary	4	N. C.	±	0	0	0	1111000000	4321000000
Dx28*	Secondary, neurorecurrence	0	N. C.	++	0	4	4	0111110000	5422100000
Ex4	Tertiary	4	9	0	0	0	0	0111110000	4321000000
Ex18*	Tabes	0	2	++++	0	3	4	1222211100	35554+21000
Ex31	Central nervous system	4	16	+++	4	4	4	1222210000	5543221000
Fx16*	Wassermann	0	2	+	0	0	0	1112210000	4321100000
Fx26	Tabes	0	2	++++	0	1	4	0112110000	4332100000
Hx5	Wassermann	4	65	++++	1	4	4	2211000000	5543210000
Hx6	Central nervous system, vascular	?	23	+++	0	0	0	1112221000	4553210000
Ix14*	Central nervous system, unclassed	4	4	++	0	4	4	1222110000	4322000000
Ix17	Secondary	4	14	+++	4	4	4	2222200000	4322000000
Ix18	Central nervous system (?), Bell's palsy	0	13	++	0	0	0	0001100000	4322000000

this type of mastic curve was the gold curve abnormal. Perhaps changes may occur in the mastic test before they appear in the gold, and in treated neurosyphilis it may be that the mastic test is rendered negative more slowly than is the gold. Further study of this type of reaction is necessary before its interpretation can be safely determined.

DISCUSSION

A general survey of the whole material shows that in 108 cases in which abnormalities appeared in one or both colloidal tests, there was complete agreement (both curves paretic in type) only thirty times. There were seventeen instances in which the gold test was positive (five paretic, twelve syphilitic zone) when the mastic test was negative. At least a few of the gold curves of the latter type may be questioned

because of possible bacterial contamination of the fluid. On the other hand, in seventeen cases the mastic curve was paretic when the gold was negative, and in all of these cases it appeared to be more closely in agreement with the clinical and serologic evidence provided. In the group of twenty-four borderline mastic curves, the evidence appears to favor the gold four times, and the mastic thirteen times, while in seven instances the result is doubtful. We must emphasize once more the relation of the paretic curves of both colloids to paresis. So far as the gold curve is concerned, practically all cases of paresis show a paretic curve, but this type of curve is frequently found in other types

TABLE 6.—GOLD CURVE OF VARIOUS TYPES; MASTIC "3" CURVE

Case No.	Diagnosis on Admission. Form of Syphilis	Blood Wassermann Reaction	Cerebrospinal Fluid					Gold Curve	Mastic Curve
			Cells	Globulin	Wassermann 0.2 C.c.	Wassermann 0.4 C.c.	Wassermann 1.0 C.c.		
P3*	Wassermann	0	N. C.	++	2	4	4	5555431000	3322110000
R5*	Tertiary	0	N. C.	0	0	0	0	1121000000	3221100000
S10*	Wassermann	4	N. C.	+	0	0	0	0112100000	3221110000
Ax5*	Tertiary	4	N. C.	±	0	0	0	1222000000	3322111000
Ax7	Central nervous system, unclassed	0	N. C.	+	0	0	0	111254*5100	3322111000
Cx7*	Tertiary	0	N. C.	±	0	0	0	0012211000	3332110000
Cx18	Central nervous system, vascular	4	N. C.	+	0	0	0	0123111000	3221000000
Cx19*	Central nervous system	0	13	+++	0	0	0	0011110000	3221000000
Cx21*	Secondary	0	N. C.	0	0	0	0	0002200000	3210000000
Cx25	Latent	0	N. C.	0	0	0	0	1121100000	3210000000
Cx26*	Wassermann	0	N. C.	0	0	0	0	112*1100000	3221000000
Cx28*	Tertiary	3	N. C.	+	0	0	0	0001000000	3221100000
Dx9	Nonsyphilitic, lethargic encephalitis	0	N. C.	+	0	0	0	1221000000	3210000000
Dx31*	Wassermann	4	8	++	0	0	0	0111100000	3210000000
Ex1*	Wassermann	4	5	++	0	0	0	1121000000	3210000000
Ex13*	Wassermann	0	3	++	0	0	0	0112000000	3221000000
Fx8*	Secondary	0	2	+	0	0	0	1211000000	3221000000
Fx12*	Secondary	0	2	++	0	0	0	11111*00000	3222000000
Fx14*	Primary	0	15	++	0	0	0	0011110000	3222000000
Gx1*	Secondary	0	3	+++	0	0	4	1111110000	3221000000
Gx3	Tertiary	4	3	±	0	0	0	111*1*1*10000	3221000000
Gx5	Wassermann	4	1	+	0	0	0	0111111000	3211000000
Gx22	Central nervous system, vascular	4	42	++	1	4	4	23*3*1100000	3221000000
Ix4*	Secondary	0	3	+	0	0	0	1110000000	3221000000

of neurosyphilitic involvement. A paretic type of mastic curve also appears in most cases of paresis, but it is found even more frequently than the gold in other neurosyphilitic diseases. A diagnosis of paresis is therefore never permissible solely on the basis of a paretic mastic curve.

We have no explanation to offer for our failure to obtain mastic curves of the syphilitic zone type, unless, as suggested by Stanton,[3] variations in the lot of mastic used is responsible. We have employed four lots of mastic from different sources, with almost equally satisfactory results in three. One lot was found to react in the usual way but not to a sufficient degree, and was therefore discarded.

We feel, however, that the information provided by the mastic test, while in our hands it does not precisely parallel that afforded by the gold test, is of value in the examination of spinal fluids of syphilitic patients. In a considerable number of cases, it has been found to be positive when the gold test was negative.

CONCLUSION

The results which we have obtained show that there is a fairly close parallelism between the colloidal gold and the colloidal mastic tests; and that when agreement is lacking, the mastic test seems to detect abnormalities more frequently than does the gold. This fact, and the simplicity of performance of the mastic test, lead us to conclude that the test should be an indispensable part of the routine of spinal fluid examinations.

DIAGNOSTIC CRITERIA IN EPIDEMIC ENCEPHALITIS AND ENCEPHALOMYELITIS

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INTRODUCTION

In this disease the variegated symptomatology, the great diversity of syndromes met with, the apparent absence of any constant chronology in the development of the syndromes, and the aping by this malady of almost every well-known and well-defined neurologic syndrome, have been responsible for many mistakes in diagnosis and have caused even some of the best-informed students of the disease almost to despair of creating any sort of order out of the clinical chaos presented. But the multifarious clinical forms are gradually being sorted and placed in a relatively small number of groups, the members of each group approximating more or less closely a certain abstract standard of characterization that is now generally recognized as one of the clinical types of the disease. And when the symptoms and signs that are recognizable by modern methods have been accumulated, and when the sites within the nervous system to which they point have been carefully determined, there is in most cases little difficulty in deciding on a diagnosis.

COLLECTION OF THE DATA ON WHICH THE DIAGNOSIS IS TO BE BASED

The Anamnesis.—This should be exact and detailed.

The prodromal symptoms that have most often been complained of include: headache, slight sore throat, chilly sensations, general weakness, disinclination for exertion, and mild gastro-intestinal upsets.

Onset and Development.—In most cases the onset of the disease is abrupt, but the initial symptoms have varied much in different epidemics. In the first Vienna epidemic there was usually a fore-stage with symptoms of slight meningeal irritation, which was soon followed by somnolence and by ophthalmoplegia. In the 1918 epidemic in Australia, there was an initial stage of excitation with headache and sometimes convulsions, and only several days later did the somnolence, the fever and the signs of paralysis appear. In the cases first observed by Netter in France (1918) symptoms of meningeal irritation were absent, but fever was more common than in Vienna. The symptomatic triad described by Netter consisted of fever, somnolence and ophthalmoplegia externa. English observers (1918) recorded asthenia,

malaise, headache, general pains, chills, nausea, anorexia and catarrhal inflammations of the respiratory tract as symptoms of the initial stage, followed in a few days, or sometimes almost at once, by any one of several clinical pictures (somnolence with ophthalmoplegia, bulbar paralysis, catatonic stupor, or, comparatively frequently, a Parkinson-like syndrome). In the United States, initial stages of various sorts have been described. When the onset was sudden, there was often severe pain in the head, fever and delirium, followed by a period of improvement for a few days, after which apathy, somnolence and cerebral nerve paralyses of different sorts or Parkinson-like syndromes developed. When the onset was more insidious, the initial symptoms often consisted of diplopia and slight mental confusion, or, in some cases of neuralgias or of pareses in the domain of one or more of the cerebral nerves; and these patients, too, often showed improvement for a few days only to become somnolent later. Not infrequently there was marked restlessness at night with insomnia before a stage of somnolence was reached. In the Hamburg epidemic of 1918-1919, described by Nonne, such an initial stage as described in the foregoing does not seem to have been marked, the disease frequently having been ushered in by paralyses of the cerebral nerves or by a Parkinson-like syndrome. In the recent epidemic in Italy, Switzerland, Austria and parts of Bohemia (1919-1920) the prodromal phenomena have consisted of anorexia, headache, vague rheumatic pains, sometimes diplopia and slight fever lasting for three or four days, then somnolence and external ophthalmoplegia, but, in addition, certain hyperkinetic phenomena (myoclonia, choreatic disturbances of motility, etc.) have been common. During the past year, hyperkinetic cases have occurred also in America. Thus, T. F. Reilly called attention to rhythmical contractions of the abdominal muscles as a common symptom; Ramsay Hunt has recently described cases of "acute infectious myoclonus multiplex"; Tilney and Howe in their monograph have reported choreiform and myoclonic types; Foster Kennedy has described them also under the caption of "acute infective neuronitis"; and Bassoe, Happ and Blackfan, and others in this country have met with choreiform and myoclonic syndromes. It would seem probable, now, that some of the cases described in France as early as 1917 as "Cruchet's disease" were instances of the hyperkinetic form of epidemic encephalitis.

Of great importance for the early diagnosis is an expert general physical, neurologic and psychiatric examination.

Somnolence.—Of the abnormal mental symptoms, somnolence or pathologic drowsiness is by far the most important as a diagnostic criterion. The peculiarities of this somnolence are now so well known

and so different, in most instances, from those of uremic or apoplectic coma that they need not be mentioned further.

Deliria.—The deliria that occur in encephalitis are less characteristic than the somnolence. In certain instances they are indistinguishable from the deliria of other toxic-infectious states (initial and febrile deliria; delirium acutum). Sometimes a delirious state assumes the characters of acute hallucinatory delirium (amentia); often the symptoms closely resemble those of delirium tremens (visual and kinesthetic hallucinations) or those of a Korsakoff's psychosis (disorientation as to time, place and persons, with pseudoreminiscences).

Paralyses.—Paralyses or pareses of the lower motor neurons, most often in those corresponding to the cerebral motor nerves giving rise to ophthalmoplegias, facial paralyses, masticatory paralysis, dysphagia or dysarthria, though occasionally also circumscribed paralyses in the trunk or in the extremities, are among the commonest disturbances of motility. Transitory diplopia, unilateral or bilateral ptosis, accommodation paralysis, sluggish pupils and strabismus are important diagnostic marks. Difficulty in reading may persist long after subsidence of the acute attack.

Spastic States.—Far less often than paralyses have there been spastic states (upper motor neuron lesions), though in a few instances hemiplegias, monoplegias or paraplegias have occurred with corresponding changes in the reflexes (ankle clonus; positive Babinski sign). Sicard (1920) has described a "paraplegic type of encephalitis lethargica."

Myostatic Disturbances.—Much more common than spastic states have been those peculiar alterations in tonus corresponding to parkinsonian rigidity, catatonic stupor and catalepsy—the so-called amyostatic syndromes—due to disturbances in the extra-pyramidal motor innervations.

Hyperkinetic Phenomena.—Signs of motor irritation (hyperkinetic phenomena) are now known to be common in these acute nonsuppurative inflammations of the nervous system. In the first Viennese epidemic hyperkinetic phenomena, other than those due to slight meningeal irritation, were uncommon; but in the later epidemics these syndromes have greatly increased in frequency, and in several they have dominated the clinical picture in a majority of the cases. Myoclonias, myokymias, fascicular twitchings, fibrillary twitchings, epileptiform convulsions general or unilateral, choreiform states and maniacal states appear to have become increasingly common.

Sensory Disturbances.—Certain disturbances of sensation can be utilized as diagnostic criteria, though the sensory side of the clinical picture is, in most cases, far less in evidence than the motor side.

Spontaneous pains—vague rheumatic pains; headache; facial, occipital, cervical, brachial, intercostal and crural neuralgias—have been fairly common and often misleading. In a few cases lancinating pains, like those of tabes dorsalis, have been noted, and in a larger number of cases multiple pains like those of polyneuritis. In the latter instances, the muscles and Valleix points have been tender on pressure. Tenderness of the masseter muscles, a sign observed repeatedly by H. M. Thomas, was demonstrable in one of my cases. In another patient who had suffered from extensive cerebral-nerve paralyses, there had been severe bilateral tic doulereux for the relief of which both trigeminal nerves had been injected with alcohol. Typical root pains are not uncommon. With Cross and Irwin I reported one such case (meningo-encephalo-myelo-neuritis) and Bassoe, of Chicago, has described root pains as a part of the meningoradicular syndrome. Boyd has observed herpes in the area of distribution of root pain. Pardee (1920) has described an acute descending radicular type of epidemic encephalitis with sharp lancinating root pains, paresthesias, muscle spasm, hyperesthesia, delirium and fever.

Anesthesias apparently have been relatively uncommon though they have been encountered in isolated cases in neural, radicular, funicular and thalamic distribution. One of my patients had hemiparesthesia with hyporeflexia of the same side.

Disturbances of the special senses have been reported, especially in the domain of the auditory nerve (cochlear and vestibular divisions), in the form of tinnitus, deafness and dizziness. The olfactory, optic, and gustatory nerves appear to have been rarely involved. The eyegrounds are usually normal, though a low grade of optic neuritis has sometimes been reported (Hiram Woods). Boyd mentions two patients who showed pronounced optic atrophy with marked failure of vision. Patients frequently complain of dimness of vision and of inability to read, but these symptoms, as a rule, have been due, less to involvement of visual conduction paths than to accommodation paralysis. The latter is fairly common and is an important diagnostic mark.

Anomalies of the Reflexes.—In most cases the tendon and superficial reflexes are not markedly altered. In some instances they have been hyperactive and, in a few, Babinski's sign has been present. In other rather rare cases, great diminution or even total abolition of the deep reflexes has been noted. The defensive reflex (reflexe d'automatisme) is often slightly increased (Guillain). Reflexes may change markedly from day to day.

Incoordination.—Disturbances of coordination have not been a marked feature in any of the epidemics, though isolated cases of ataxia,

most often of the cerebellar type, have been observed. Aphasia, agraphia and apraxia have not been common. Nystagmoid movements of the eyes were observed in five of seven cases by Hiram Woods (1919), and in two these were the only eye-movement disturbances demonstrable.

Autonomic Disturbances.—In autonomic domains some important disturbances have been noted. Alterations in the pupils and in the pupillary reactions have been found to be exceedingly common, and they are now ranked among the more important diagnostic signs. Inequality of the pupils (anisocoria), irregular-shaped pupils, sluggish pupillary reflexes, definite Argyll Robertson pupil (unilateral or bilateral), and total pupillary rigidity have been encountered. Accommodation-spasm and particularly accommodation paralysis have been common. Spasm of the sphincter iridis muscle has also been seen. W. B. Cadwalader (1920) has reported "bilateral sympathetic ophthalmoplegia." This bilateral Horner syndrome consists of: (1) contraction of the pupil, (2) narrowing of the palpebral fissure, and (3) slight retraction of the eyeball. He thinks the bilateral syndrome must depend on diffuse involvement of the brain stem. Salivation has been a frequent symptom. In some instances it has depended on jaw-drop; in others it has resulted from greatly increased secretion. Tachypnea, tachycardia, esophagospasm, esophageal dilatation, neurointestinal disturbance, retention of urine, vasomotor disturbance in the skin, and hyperhidrosis are among the autonomic symptoms that have been most frequently recorded. Trophic disturbances (other than muscular atrophies) most worthy of emphasis are a tendency to rapid emaciation and, in the very severe cases, to decubitus. In a few patients a peculiar swelling of the face suggestive of myxedema has been observed. Herpetic eruptions, particularly herpes labialis, have been common, especially in the initial stage. In a few cases scleroderma has occurred as a sequel. Peculiar exanthems (petechial, roseolar, vesicular) have occasionally been seen, but they appear not to have been common. Whether these represent trophic disturbances, or as would seem more probable, localizations of the virus in the cutaneous capillaries, we do not know.

Cerebrospinal Fluid and Nasopharyngeal Washings.—Much attention has been paid to examination of the cerebrospinal fluid. In the early European epidemics many reported negative findings, and a negative spinal fluid was at first regarded as characteristic. William Boyd, who studied the Winnipeg epidemic, states that "a normal fluid was the rule rather than the exception"; in a number of instances he observed increased pressure, slight pleocytosis and a slightly positive globulin reaction. In the United States, though many negative fluids

have been reported, there have been a large number in which slight increase in the cell count (small mononuclears), a positive globulin reaction, and a negative Wassermann reaction have been found. When the cell count is increased it is, as a rule, not markedly increased. Counts of from 10 to 100 are much commoner than larger numbers. The cell count may vary materially from time to time in the single case. Thus, in one of our cases it was 97 on the twenty-first day, 133 on the twenty-sixth day and 33 on the seventy-fifth day. Similar observations have been made by Wegeforth and Ayer and by Marie and Mestrezat (1920). In isolated instances counts of several hundred cells or even of several thousand cells have been made; in these cases great care must be taken to rule out the Heine-Medin disease and tuberculous meningitis. No film forms in the cerebrospinal fluid in epidemic encephalitis. A notable amount of sugar, a sign known as hyperglycorachia, has been described (Netter; Dopter). It is not accompanied by hyperglycemia.

As a rule, the cerebrospinal fluid is colorless, though occasionally it is yellowish as in Froin's syndrome. William Hough and H. H. Kerr of Washington have made one such observation. The fluid may or may not be under increased pressure. An interesting observation, frequently made, has been the temporary disappearance of the somnolence and marked relief of the irritation symptoms after lumbar puncture.

Meningococci, pneumococci, influenza bacilli and tubercle bacilli have been uniformly absent from the cerebrospinal fluid. In Austria and in Italy it is asserted that von Wiesner's *Diplostreptococcus pleomorphus* has sometimes been obtained from the fluid and also from the blood. In this country, Loewe and Strauss report that they have demonstrated their filtrable virus in the cerebrospinal fluid as well as in nasopharyngeal washings, and they regard especially animal inoculation with nasopharyngeal washings as an important diagnostic aid in the more obscure cases. They report that they have grown this filtrable virus by making use of Noguchi's method under anaerobic conditions. Levaditi and Harvier have confirmed several of Loewe and Strauss' experiments.

Leukocytosis.—The leukocyte count of the blood is of relatively little help as a diagnostic criterion. In the acute stage there is usually moderate leukocytosis, but in many of the milder cases the leukocyte count has not been increased. In initial stages suggestive of typhoid fever, however, a polymorphonuclear leukocytosis rather than a leukopenia favors the diagnosis of encephalitis.

Urine.—The urine may be entirely normal, though in the severer cases with high fever there is nearly always a trace of albumin and a few casts.

Acuity.—Most of the patients have presented an acutely developing syndrome, but there are also many subacute cases and some remarkable chronic cases with recrudescences and relapses extending over months.

There have also been a number of peracute cases with foudroyant syndromes (encephalitis siderans) terminating fatally within twenty-four or forty-eight hours from acute intoxication. The symptoms are general rather than focal and appear to be of toxic rather than of inflammatory infiltrative origin. The virus, whatever it is, can not only gradually give rise to local areas of inflammation and infiltration widely disseminated throughout the central and peripheral nervous system, but also to intoxication of the body as a whole, causing parenchymatous degenerations of the nervous system and of the viscera (von Economo). If this conception of the pathogenesis of the disease should prove to be correct, we shall be helped by it to understand some of the peculiarities of the symptomatology.

CLINICAL TYPES OR STANDARDS

Out of the welter of symptoms and signs of this protean disease, clinicians have been busily engaged in setting up clinical types or standard symptom-groups. McNalty in England, Bassoe, House, Tucker, Tilney and Howe and others in America, Netter, Sicard, Guillain and others in France, Reichart, Siemerling, Nonne and Naef in Germany, von Economo and Dimitz in Austria and Sabatini in Italy, are among those who have made notable contributions to classification.

Some classifications are based on the mode of onset and course (peracute, acute, subacute, chronic), some on intensity or severity (fatal, severe, medium, mild, abortive, rudimentary, ambulatory), some on the supposed predominant anatomic localization of the process—hence the terms encephalitis, meningo-encephalitis, mesencephalitis, poliencephalitis superior, poliencephalitis inferior, bulbar myelitis, encephalomyelitis, poliomyelitis, polyneuritis, and meningo-encephalo-myeloneuritis. Another classification is based on the supposed cause of the symptoms (toxic encephalitis, inflammatory infiltrative encephalitis). From the more purely clinical standpoint, based on symptomatology, it is desirable to select certain special forms or syndromes as types, making free use of all the classifications thus far put forward. It may be useful to adopt, for the present, the following dominant clinical types, understanding however, first, that in a single case two or more of these types may be observed, either simultaneously or successively, and, second, that as knowledge grows, it may become desirable to change the number:

1. The somnolent-ophthalmoplegic type (febrile or afebrile).

2. The paralytic (akinetic or hypokinetic) type.

3. The amyostatic type (Parkinson-like and cataleptic syndromes).

4. The hyperkinetic type (myoclonic forms, choreatic forms, epileptic forms).

5. The psychotic type (delirious, maniacal, depressive).

6. The hyperalgesic type (painful forms).

7. The tabetic type (Argyll Robertson pupils with loss of deep reflexes and, sometimes, lancinating pains).

8. The ataxic type.

9. The abortive type (formes frustes; imperfect, rudimentary, and ambulatory forms).

10. The aberrant type (intestinal forms, cutaneous forms, vagal forms, etc.).

The names are brief symbols by means of which we eiptomize in each instance a group of symptomatic details. They are vastly useful for the saving of time and for economy of thought. The occurrence of any one of these syndromes at a time when encephalitis is epidemic, especially when it is associated with signs of infection (fever or leukocytosis), should make one think of the possible existence of the disease.

THE CRITERIA FOR DETERMINING THE SITE OF THE LESIONS IN EPIDEMIC ENCEPHALITIS

Many of the syndromes mentioned are now known to be due definitely to certain focal lesions. These nonsuppurative inflammations are widely disseminated pathologic-anatomic processes, but there are certain sites of predilection, and it is because of this that certain clinical syndromes are prone to occur with especial frequency.

The site of greatest predilection in this really terrible disease is the so-called brain stem and especially the gray matter about the aqueductus cerebri and in the floor of the fourth ventricle. Other sites of predilection include: the corpus striatum, including (*a*) the nucleus lentiformis, made up of the globus pallidus and the putamen, and (*b*) the nucleus candatus; the thalamus and the hypothalamic region; the gray matter of the spinal cord, especially of its anterior horns, and the leptomeninges. But foci may occur in any part of the central nervous axis or of the peripheral nerves, including the ganglions on the sensory roots of the cerebrospinal nerves and the sympathetic system.

The limits of this article will not permit a full discussion of the diagnostic criteria for the localization of the lesions responsible for all the disturbances encountered in the disease, but a few of the more interesting points may be referred to.

The somnolent ophthalmoplegic syndrome points, of course, to the interbrain and the midbrain particularly. The somnolence itself probably depends on lesions of the gray matter about the aqueductus cerebri (centrale Höhlengrau), though just what the mechanism is has been a matter of dispute. C. K. Russel (1920) suggests that slight swelling might obstruct the aqueduct and cause acute hydrocephalus. The ophthalmoplegia directs attention to the nuclei of the third, fourth and sixth nerves. The fleeting signs are probably due to intoxication or edema, the more permanent ones to inflammatory infiltration or to toxic degenerative change.

The akinetic and hypokinetic syndromes (other than ophthalmoplegia) are usually easily localizable, for they also are in the main dependent on lesions of the gray matter containing the nuclei of origin of the corresponding motor nerves, though in some instances, the motor white tracts may be involved or even the roots of the peripheral nerves or these nerves themselves. Facial paralysis, abducens paralysis, and paralysis of the muscles of mastication, point to the pons. When these paralyses exist, the picture is that of poliencephalitis superior acuta. When there is paralysis of the muscles of deglutition (dysphagia) or of the muscles of articulation (dysarthria), often combined with disturbances of respiration and of the circulation (dyspnea, tachypnea, tachycardia), the symptoms point to the motor nuclei of the medulla oblongata. Here we have the well-known picture of poliencephalitis, inferior acuta, or bulbar myelitis. Not infrequently such paralytic phenomena are associated with tinnitus, deafness or dizziness (due to lesions of the cochlear and vestibular components of the nuclei terminales nervi acustici). Paralyses of the spinal nerves point to inflammation of the anterior horns of the spinal cord.

When hemiplegias and monoplegias occur with spastic phenomena and a positive Babinski sign, the lesions are most often located in the cerebrum (cortical, capsular or peduncular). Occasionally a pyramidal tract lesion with a positive Babinski sign may be located below the cerebrum (in the pons, the medulla oblongata or the spinal cord). Paraplegia in this disease is most often due to a transverse lesion of the cord, though it may occasionally depend on extensive bilateral cerebral lesions.

The amyostatic syndromes (including the Parkinson-like syndrome and cataleptic rigidity) are most interesting as localizing guides. The picture of paralysis agitans sine agitatione is of frequent occurrence in epidemic encephalitis, and cataleptic rigidity is by no means uncommon. The studies of S. A. K. Wilson, of von Strümpell, of Ramsay Hunt and of others have made it probable that these myostatic disturbances depend on injury to the corpus striatum and especially to the globus pallidus of the nucleus lentiformis.

The several hyperkinetic syndromes may also be valued to a certain extent for localizing purposes. The motor irritation of the initial stage may depend on the general intoxication or on meningeal irritation. Fibrillary and fascicular twitchings point to the cell bodies of the lower motor neurons. Rhythmic myoclonic contractions probably point to supranuclear irritation, though it is possible that some of the myoclonias depend on direct or reflex irritation of the lower motor neurons. Hamill (1920) assumes that the bilaterally symmetrical rhythmical movements point to some mechanism or center in the medulla oblongata. His tracings indicated that the clonic contractions of muscles that come into play in forced respiration had a definite relation to the respiratory rhythm. The rate of twitching equaled the respiratory rate or was double this rate. Choreatic disturbances of motility point to the encephalon, probably to the diencephalon rather than to the telencephalon, judging from what we know of the posthemiplegic choreas and of the thalamic syndrome. It is conceivable that a chorea may be due to diffuse intoxication of the encephalon rather than to localized lesions in small areas, though of this we are not certain. It is possible too that myoclonic syndromes, like choreatic and athetotic disturbances of motility, may depend on the removal of inhibitory influences through injury to their centers of origin. Epileptiform seizures, especially when of the jacksonian type, point to irritation of the cortex cerebri.

The psychotic types doubtless depend on diffuse toxic disturbances or multiple foci of infiltration in the telencephalon, but we know too little regarding the localization of psychic functions to permit a more definite statement. When abnormal mental states persist after the initial stage, one can be tolerably sure that a telencephalitic process or a meningo-encephalitic process exists. This would seem especially probable in patients who exhibit pronounced amnesias, presenting the picture of senile dementia or that of dementia paralytica.

To a certain extent the hyperalgesic states can also be utilized for purposes of localization. Multiple pains of neural and radicular distribution point to multiple neuritis or radiculitis. Typical zone-like pains extending transversely around the trunk, or vertically along the extremities, point to their respective nerve roots (radiculitis) or to foci in the posterior horns of the spinal cord (poliomyelitis posterior). Hemi-anesthesia with violent persistent pains on the anesthetic side, especially with other components of the thalamic syndrome described by Déjerine and Roussy, point to the ventrolateral region of the contralateral thalamus. Howe of New York has reported an example of the thalamic syndrome caused by epidemic encephalitis.

Visual or auditory hallucinations point to irritations of the cuneus or of the first temporal gyrus, respectively, though irritative lesions in

the visual and auditory corticopetal conduction paths might be responsible.

Ataxia of the cerebellar type, with defective coordination of agonists, antagonists and synergists, points to the cerebellum, or to the paths connecting the cerebellum with other centers (vestibular nuclei, red nuclei, etc.). Hemi-ataxia can result from injury to the cerebello-thalamic or rubrothalamic paths on one side, or it may follow on lesions of the lemniscus medialis.

The interesting tabetic syndromes now being occasionally reported probably point to an intoxication, injuring the posterior funiculi of the spinal cord and the light-reflex arc somewhere in the midbrain.

Retention of urine and other autonomic disturbances give opportunity for interesting localizing speculation, but we are as yet poorly informed regarding their anatomic basis. Much of what I have said of the localization of the encephalomyelitic foci is based on deductions from our general knowledge of localizing diagnosis rather than on actual pathologic-anatomic studies of necropsy materials from cases of encephalitis that have been well studied clinically.

A fair classification is possible based on the sites of the toxic-infectious processes in encephalitis. The term encephalitis would include: (1) all the inflammatory processes from the telencephalon to the mesencephalon inclusive (telencephalitis, diencephalitis, mesencephalitis), these together making up cerebral encephalitis, and, in addition, (2) all the inflammations of the rhombencephalon (pons and medulla oblongata), including pontile encephalitis and bulbar myelitis. Under the term myelitis would be included all the inflammations that occur in the spinal cord (poliomyelitis anterior, poliomyelitis posterior, myelitis transversa, and myelitis funicularis). Under neuritis could be included the inflammations that occur in the extracentral portions of the cerebral and spinal nerves (radiculitis, peripheral neuritis). Under meningitis, or rather leptomeningitis, would be included patchy inflammations of the soft meninges. Various combinations of these different local inflammations occur in a single case. In some instances it would seem legitimate to speak even of a meningo-encephalo-myelo-neuritis.

DIFFERENTIATION OF EPIDEMIC ENCEPHALITIS FROM SIMULATING INFECTIONS AND INTOXICATIONS

Among the infectious processes with which epidemic encephalitis is likely to be confused stand out prominently: (1) meningitis, (2) influenza and grippal infections, (3) the Heine-Medin disease, and (4) multiple neuritis, especially the infectious form. Confusion may also occur, though less frequently, with (5) typhoid fever, (6) mumps, (7) infectious arthritis or myositis, (8) tetanus, or (9) hydrophobia.

Here, too, should be considered the differentiation from (10) forms of encephalitis, myelitis or encephalomyelitis other than those due to the specific virus that must be responsible for the present epidemic.

Among intoxications that may closely resemble epidemic encephalitis with somnolence may be mentioned: (1) uremia, (2) acidosis, (3) cholemia, (4) drug intoxications, and (5) botulism.

Meningitis.—Early or late in the course of epidemic encephalitis, symptoms of meningeal irritation (general hyperesthesia, rigidity or retraction of the neck, convulsive seizures, photophobia, pupillary disturbances, eye-muscle paralyses, Kernig's sign) not infrequently occur. Indeed, the leptomeninges are now known to show patchy infiltration with small mononuclear cells and sometimes a few polymorphonuclear cells. One of the surprises in these cases, however, is the paucity of changes in the cerebrospinal fluid as compared with the pathologic-histologic findings and sometimes with the clinical phenomena. In many cases, even in those exhibiting signs of meningeal irritation, the spinal fluid may be quite normal, or may show only a faintly positive globulin test or a little increase of sugar. In a certain proportion of the cases, however, the cell count is a little increased, the cells numbering from 10 to 50, chiefly small mononuclears, and the globulin test is distinctly positive. In rare instances a high cell count is encountered. The fluid may or may not be under increased pressure. No film forms in it. The fluid as a rule is colorless, though it may be tinged slightly yellow, or may contain now and then a few red corpuscles.

In epidemic cerebrospinal meningitis the spinal fluid nearly always differentiates the condition from epidemic encephalitis. The fluid is distinctly turbid; there is marked pleocytosis with predominance of polymorphonuclear neutrophils, and meningococci are present in stained smears, either free or inside the cells.

In the forms of purulent meningitis other than epidemic cerebrospinal meningitis, the fluid is turbid; there are many pus cells, and the causal bacteria (pneumococci, streptococci, influenza bacilli, typhoid bacilli) are usually demonstrable in smears, cultures, or by animal experiment.

In the meningitic form of the Heine-Medin disease, the cell count in the cerebrospinal fluid is usually much higher than in epidemic encephalitis.

Mistakes in diagnosis most often occur in the differentiation between epidemic encephalitis and tuberculous meningitis. A number of cases have been diagnosed as epidemic encephalitis only to turn out to be tuberculous meningitis, and the reverse has also been true. Interesting discussions of this confusion are to be found in the papers of Loygue (1919) and of Pirie (1919). The onset is usually more acute in epi-

demic encephalitis than in tuberculous meningitis, but in some cases there is an insidious onset of the encephalitic symptoms in which event one may easily be misled, especially when a young person is attacked. But in tuberculous meningitis the fluid is often (though not always) turbid; the cell count is relatively high, even though small mononuclear elements may predominate; a film usually forms in the fluid, and quite frequently, though not always, tubercle bacilli can be demonstrated in the centrifugate when a number of specimens are studied. Moreover, the rigidity of the neck and Kernig's sign are usually much more pronounced in tuberculous meningitis. The control of diagnosis by necropsy has shown, however, that the two diseases are still occasionally confused even when the most careful studies are made.

Influenza and Grip.—Many patients who develop encephalitis exhibit the signs of the disease during or shortly after suffering from an infection that has been diagnosed sometimes as influenza and sometimes as la grippe. In some of these cases the infection may, from the beginning, have been that of acute epidemic encephalitis; in other cases, undoubtedly influenza or la grippe has preceded the epidemic encephalitis. We know, too, that influenza and grippal infections other than the Spanish influenza are capable of causing encephalitis as a complication. There is every reason to believe that the infectious agent of the grip group of diseases can localize in the meninges and brain and give rise to metastatic encephalitis. The confusion at present is great because many believe that acute epidemic encephalitis itself is merely a nervous form of grip. Though the evidence now is against this, the whole matter is decidedly unsettled.

Heine-Medin Disease.—Aside from the meningitic form of the Heine-Medin disease, there are still other forms that may be mistaken for one or another form of epidemic encephalitis. We know now that the Heine-Medin disease, besides producing poliomyelitis, is capable also of producing multiple neuritis, meningitis, poliencephalitis, cerebritis and cerebellitis. We know also that acute epidemic encephalitis, though most often giving rise to symptoms of poliencephalitis, diencephalitis or cerebritis, may also give rise to characteristic poliomyelitis pictures or polyneuritic syndromes. Indeed, the outbreak in Australia, described by Breinl in 1918, with its hyperkinetic phenomena and poliomyelitic syndromes, was supposed at the time to be a peculiar form of the Heine-Medin disease. George Draper, in his studies of the English outbreak of epidemic encephalitis, kept strongly in mind the possibility that the disease might be due to the virus of the Heine-Medin malady. In the Italian and Austrian epidemics of 1919, poliomyelitic pictures were often encountered. In the United States, too, several

observers have described poliomyelitic forms of acute epidemic encephalitis.

Until we are sure of the exact nature of the virus in the Heine-Medin disease on the one hand and in epidemic encephalitis on the other and can determine their presence in patients, a final decision as to the identity or nonidentity of the two viruses cannot be reached. In the meantime, it would seem best to assume that they are two entirely different diseases, even though we concede the possibility that the one virus may be a modification of the other. Loewe and Strauss find that injection into animals of the cerebrospinal fluid from patients suffering from encephalitis will reproduce the disease. This is not true of the Heine-Medin disease. One attack of the Heine-Medin disease appears to yield permanent immunity, and Boyd has observed the occurrence of epidemic encephalitis in one patient who had had poliomyelitis in earlier life. The diagnostic criteria relied on at present are:

1. In the Heine-Medin disease the paralyses are more or less complete, many are permanent and they are usually present from the beginning, whereas in epidemic encephalitis the paralyses are more likely to come on slowly and to involve one muscle or one group of muscles after another, to be partial rather than complete and to be transient, the muscles later usually recovering their power if the patient does not die.

2. Unilateral or asymmetrical paralyses are common in the Heine-Medin disease, whereas bilateral symmetry of the paralyses is more often encountered in encephalitis.

3. The fever is as a rule highest before the paralysis occurs in the Heine-Medin disease; in encephalitis the fever is usually slight and may be more marked after paralysis has occurred.

4. In the Heine-Medin disease leukocytosis is more marked than in epidemic encephalitis.

5. The cell count in the cerebrospinal fluid is much higher in the Heine-Medin disease.

6. Children and adolescents are more often affected by the Heine-Medin disease than persons of other ages; epidemic encephalitis attacks persons of all ages.

7. The distribution of the typical cases is different in the Heine-Medin disease than in epidemic encephalitis when the two maladies are epidemic at the same time in a given country.

8. In epidemics of the Heine-Medin disease the encephalitic and bulbar-paralytic types are relatively uncommon; in epidemics of lethargic encephalitis the poliomyelitic syndromes are relatively uncommon.

Multiple Neuritis.—There is some evidence that the peripheral nerves may actually be involved in some cases of encephalitis. It is rare, however, even in these cases that symptoms and signs pointing to involvement of the nerve centers are lacking. Furthermore, the paralyses that occur in encephalitis differ in their distribution from those of multiple neuritis. The chronologic sequence is also different, since in encephalitis it is common to have first eye-muscle paralyses, later facial paralysis and masticatory paralysis, then bulbar paralysis and lastly paralyses of the extremities; the reverse order occasionally occurs, but the former is the rule. It would be rare to see such a sequence of events in multiple neuritis. Moreover, ophthalmoplegia externa and sphincter disturbances (bladder and rectum), common enough in encephalitis, are rare in neuritis.

Encephalitis in which there are accommodation paralysis and paralysis of the muscles of deglutition, so that fluids return through the nose, may excite suspicion of diphtheritic paralysis, but in the latter condition evidences of pahryngeal, nasal or laryngeal diphtheria should be present; smears or cultures should yield the Klebs-Loeffler bacillus, and the glands at the angle of the jaw should be enlarged.

Typhoid Fever.—When epidemic encephalitis broke out in this country there were a number of cases supposed to be typhoid. A similar confusion has been reported in other countries. However, a negative blood culture in the first week, later, negative Widal reaction, leukocytosis instead of leukopenia, absence of acute splenic tumor and roseola, presence of paralyses and of hyperkinetic phenomena will exclude typhoid.

Mumps.—It is known that ordinary mumps may be complicated by an encephalitis and that acute parotitis may complicate epidemic encephalitis, since several examples of this have been reported by Lesné and Langle (1920). One observer in this country has described cases presenting the clinical picture of acute epidemic encephalitis (without any complicating parotitis) as mumps-encephalitis because they occurred coincidently with an epidemic of mumps.

Certain clinical features of mumps are helpful in the differential diagnosis from epidemic encephalitis complicated by parotitis. Thus, mumps is extremely contagious from person to person, whereas the contagiosity of epidemic encephalitis seems to be slight. Again, mumps does not occur a second time in patients who have once had the disease, whereas a majority of the patients, who suffer from epidemic encephalitis have perhaps earlier in life suffered from mumps. Pathologic drowsiness, ophthalmoplegia and other paralyses do not occur in mumps unless the mumps be complicated by encephalitis.

Infectious Arthritis and Myositis.—In some of the milder cases of encephalitis the symptoms chiefly complained of are pains in the neck, shoulders and back, or in the extremities, sometimes leading one to think of infectious arthritis or myositis. Careful clinical and roentgen-ray examination, consideration of the mode of onset, a history of pathologic drowsiness, the discovery of slight ophthalmoplegia, of other paralyses or of pupillary disturbances and examination of the cerebrospinal fluid, will usually differentiate.

Tetanus.—In a few instances the diagnosis of tetanus has been made in cases of epidemic encephalitis. Though in encephalitis paralysis of the jaw muscles is commoner than trismus, irritation of the motor nucleus of the nervus trigeminus in encephalitis can give rise to definite trismus. If this be associated with marked rigidity of the neck and spine, one could easily be misled. But in tetanus there is usually (1) a history of a contaminated penetrating wound; (2) fever is not present at first and does not appear until just before death, whereas in encephalitis elevation of temperature is not infrequent at the beginning; and (3) pathologic drowsiness and ophthalmoplegias are absent.

Hydrophobia.—In isolated instances epidemic encephalitis has been confused with hydrophobia or rabies, since in both there may be a hyperkinetic early stage followed by a paralytic stage (with paralysis of the face, tongue, eye muscles and extremities). But rabies is preceded by a history of bite by a dog or other animal, and it is customary when a human being has been so bitten to determine by inoculation of a rabbit or by examination for the Negri bodies or for lesions of Van Gehuchten and Nelis, whether or not rabies actually exists in the animal. Epidemic encephalitis might of course develop in a person who had been bitten by an animal, in which event the differential diagnosis might be difficult.

Forms of Encephalitis, Myelitis and Encephalomyelitis Other Than the Epidemic or Lethargic Form.—Epidemics of encephalitis and of encephalomyelitis have undoubtedly occurred before, but whether they were due to the same virus as the epidemic encephalitis now prevailing is not known. Some investigators appear to believe that the disease now prevailing is entirely new. Others, including myself, think it probable that we are not dealing with a new disease, but with one that caused epidemics in earlier times. Sporadic cases of encephalitis have long been known to occur. That there are many forms of encephalitis due to different viruses (bacterial and other), there can be no doubt. Can we distinguish these other forms of encephalitis and encephalomyelitis from the acute epidemic or lethargic encephalitis now prevailing?

The poliencephalitis superior acuta of Wernicke has long been known to be a hemorrhagic inflammation of the pons occurring most often in drunkards. The acute hemorrhagic encephalitis described by Strümpell and later by Leichtenstern, though it caused clinical syndromes closely resembling some of those observed in the present epidemic, seemed more often to give rise to hemiplegia, monoplegia and aphasia through massive involvement of the telencephalon. The lesions, besides being more massive were much more hemorrhagic in character than has been commonly observed in the present epidemic. It does not seem to me, however, at all certain that either Wernicke's encephalitis or the Strümpell-Leichtenstern encephalitis is fundamentally different from the encephalitis now prevailing. They may possibly have been caused by the same virus or by a close relative. Perhaps it is better, provisionally, to regard them as entirely separate diseases. In the meantime, no wholly satisfactory method of clinical differentiation is available.

Uremia.—In several cases of epidemic encephalitis in my own practice, the patients had been sent to me with a probable diagnosis of uremia, on account of the pathologic drowsiness, associated with albumin and casts in the urine and, sometimes, with arterial hypertension. But the coma of uremia is much deeper than that of epidemic encephalitis; the phthalein output is markedly reduced in uremia and the blood nitrogen greatly increased. Moreover, in uremia the cerebrospinal fluid yields negative findings. In epidemic encephalitis the cerebrospinal fluid may, it is true, be normal, but often there is a slight increase in the count of small mononuclear cells and a positive globulin reaction. It is rare, too, in uremia to encounter the types of paralysis, of hyperkinesis and of myostatic disturbance that are common in epidemic encephalitis.

Acidosis.—Diabetic coma with acidosis may occasionally be confounded with epidemic encephalitis, especially if the patient with encephalitis has undergone starvation and has a trace of sugar in the urine (not uncommon). The mistake should, however, rarely be made, for diabetic coma is always, or nearly always, preceded by a long history of diabetes mellitus, whereas the onset of epidemic encephalitis is, as a rule, acute in patients who do not have glycosuria.

Cholemia.—Here, too, confusion will be rare, the history of preceding hepatic disease and the mode of onset of the cerebral symptoms helping in the differentiation.

Drug Intoxications.—Acute veronal poisoning, cocain poisoning and other forms of drug intoxication might at times of prevalence of epidemic encephalitis lead through drowsiness or delirium to diagnostic

mistakes. A drug delirium usually sets in abruptly with confusion and hallucinations. Changes in the reflexes, pupillary anomalies and dysarthria are not uncommon.

A case of veronal (barbitol) poisoning simulating epidemic encephalitis has been reported by Hassin and Wien of Chicago (1920). The patient took 30, 60 and 90 grains, respectively, on three occasions, the ingestion being followed by profound sleep from which she could be aroused to talk coherently but sluggishly. She would fall asleep again during conversation. She had fever, diplopia and asthenia. Often a history of drug addiction is available and will call the physician's attention to possible intoxication of this sort.

Alcoholic Psychosis.—Acute alcoholism with its coma, delirium tremens with its dreamy states, abundant hallucinations, tremor, fever and tachycardia or Korsakoff's psychosis with its disorientation as to time, place and persons, and its memory falsifications might in some instances be mistaken for epidemic encephalitis, which is now known to be capable of causing somewhat similar syndromes. Moreover, encephalitis frequently occurs in patients addicted to alcohol.

Botulism.—In the epidemic that prevailed in Vienna in 1917, the patients were at first supposed to be suffering from sausage poisoning, and in the epidemic in England in 1918 many of the cases of encephalitis were first reported as instances of food poisoning or botulism. In this country, too, epidemic encephalitis was occurring at a time when outbreaks of true botulism due to poisoning from ripe olives were occurring.

At the time of the great war, the use of canned foods (meat, fish, lobster, sausage, mushrooms, olives) was greatly increased all over the world, and opportunities for food poisoning were multiplied. Botulism causes bulbar paralysis and thus simulates the pontile and bulbar-myelitic forms of encephalitis. It is not surprising that confusion of botulism with epidemic encephalitis should have occurred.

In botulism, there is usually bilateral ophthalmoplegia of the intrinsic muscles of the eye, and though this may occur also in epidemic encephalitis, ophthalmoplegia externa is much more common. When botulism occurs *Bacillus botulinus* can usually be demonstrated in the food that has caused the poisoning. Moreover, the persons who become ill are members of groups that have partaken of the contaminated food.

DIFFERENTIATION OF EPIDEMIC ENCEPHALITIS FROM SIMULATING STATES DUE TO VASCULAR LESIONS

The vascular lesions most likely to give rise to confusion with epidemic encephalitis are: (1) cerebral hemorrhage, (2) cerebral thrombosis, (3) cerebral embolism, (4) sinus thrombosis, and (5) cerebral atherosclerosis.

Cerebral Hemorrhage (Apoplexy).—Epidemic encephalitis attacking a person with arterial disease may lead to error. A true apoplectiform onset is, however, uncommon in encephalitis. Further, though hemiplegias, monoplegias and aphasias may be due to encephalitis, they have been uncommon clinical findings in the present epidemic. Moreover, at the onset of apoplexy there is no fever, though a little fever may develop later. With cerebral hemorrhage there is as a rule deep coma, so that it is impossible to arouse the patient by external stimuli, a very different condition from the somnolence of epidemic encephalitis.

Hemorrhage into the pons or medulla oblongata occurring in patients past middle life may, however, cause real difficulties in diagnosis, though gradual extension of the paralyses downward from the eye-muscle nuclei, such as is often seen in encephalitis, is rare in mesencephalic or pontile hemorrhage due to rupture of a blood vessel.

Cerebral Thrombosis with Encephalomalacia.—If a patient has atherosclerosis with low blood pressure (decrescent form of atherosclerosis), cerebral softening may be suspected when in reality encephalitis exists. The presence of fever, the mode of onset and the course of the process will usually serve as clues for differentiation.

Cerebral Embolism.—In patients suffering from a mitral lesion, cerebral embolism might be suspected when the symptoms of encephalitis appear. In encephalitis, however, the symptoms appear less abruptly and are rarely so unifocal in nature as in cerebral embolism. In the pontile and bulbar forms of paralysis one should, however, always consider the possibility of occlusion of a branch of the basilar or vertebral artery.

Sinus Thrombosis.—The autochthonous form of sinus thrombosis that is encountered, especially in chlorotic patients, may closely simulate encephalitis. If there are no signs of stasis in the veins of the skull or face, differentiation may be difficult or impossible, especially if the process is acute and soon fatal. A protracted course indicates encephalitis.

Cerebral Atherosclerosis.—In addition to cerebral hemorrhage or thrombosis there are cases of cerebral atherosclerosis without signs of apoplexy or of sudden arterial occlusion in which cerebral symptoms appear (mental deterioration, restlessness, transitory paralyses, transitory aphasias, etc.) that might be confounded with atypical and afebrile encephalitis. Von Economo has described a remittent encephalitis that during life gave symptoms of pseudobulbar paralysis with athetosis; at necropsy lesions of encephalitis (recent and earlier) were demonstrable. Sometimes examination of the cerebrospinal fluid will differentiate, but only too often the fluid is negative in encephalitis.

DIFFERENTIATION OF EPIDEMIC ENCEPHALITIS FROM SIMULATING STATES DUE TO SYPHILITIC OR PARASYPHILITIC PROCESSES

Cerebrospinal syphilis, dementia paralytica and tabes dorsalis are sometimes believed to exist when the patient's condition is due to epidemic encephalitis. Epidemic encephalitis may occur in patients with cerebrospinal syphilis, in which event one can make sure that syphilis exists, but one may have difficulty in proving the coexistence of epidemic encephalitis. These difficulties have been discussed by Jeanselme (1920).

Cerebrospinal Syphilis.—Epidemic encephalitis can give rise to symptoms that may easily be mistaken for a basilar gummatous meningitis (cerebral nerve paralyses) or for syphilitic meningo-arteritis (convulsive seizures, hemiplegia, aphasia, fever), but if the Wassermann reaction in the blood and in the cerebrospinal fluid is entirely negative, syphilis can be ruled out. Moreover, the optic nerves and optic tracts are often involved in syphilis, rarely in epidemic encephalitis. Oscillation of the symptoms may occur in either disease, though it is more pronounced perhaps in syphilis.

Dementia Paralytica.—I have seen two patients who exhibited marked mental deterioration, loss of memory for recent events, facial paresis and trembling, inability to calculate and change in behavior that led even skilled neurologists and psychiatrists to make a probable diagnosis of dementia paralytica, but in both patients the Wassermann reaction was negative both for the blood and cerebrospinal fluid, and a careful anamnesis revealed a period of pathologic drowsiness and of other symptoms that made an encephalitic origin for the symptoms seem probable.

Tabes Dorsalis.—Encephalitic cases are now occurring in which there are sluggish pupils, or even typical Argyll Robertson pupils (unilateral or bilateral) with or without abolition of the deep reflexes and lancinating pains, cases that a few years ago we should have had no hesitation in pronouncing tabes. But the Wassermann reaction in the blood and in the cerebrospinal fluid is negative and there is no syphilitic history. Cases of this sort were described in Munich by Naef in 1918, and in Hamburg, in 1920, by Nonne, and I have observed two instances.

From now on we shall have to be careful in our valuation of the Argyll Robertson pupil, which we have always regarded as of syphilogenous origin. Cases are now being reported in which the Argyll Robertson pupil has remained as a residual long after the other symptoms of encephalitis have cleared up.

DIFFERENTIATION OF EPIDEMIC ENCEPHALITIS FROM SIMULATING SYNDROMES DUE TO INTRACRANIAL NEOPLASM

Epidemic encephalitis may occasionally give rise to clinical pictures that suggest cerebral tumor, cerebellar tumor, pontile tumor or tumor of the cerebellopontile angle. Tumors of the pons, as is well known, may remain latent for a time and then become acutely manifest.

Lethargic encephalitis may, in all probability, cause lesions of the acoustic nerve with tinnitus, deafness and vertiginous attacks that lead to the suspicion of labyrinthine disease, of an acousticus tumor, of a cerebellar tumor or of a circumscribed meningitis serosa in the lateral recess (Bárány syndrome).

Epidemic encephalitis attacking the cerebellum or the cerebellar pathways and giving rise to acute cerebellar ataxia may excite suspicion of a cerebellar tumor. The course of the disease should soon remove doubt. In brain tumor, the symptoms develop gradually, as a rule, and are, on the whole, steadily progressive over a long period. In cerebellar tumor choked disk is a prominent and early symptom, whereas in epidemic encephalitis, choked disk is rarely if ever seen, and even low grades of optic neuritis are not common.

In tumors of the cerebellopontile angle, the exact chronologic development of the symptoms is characteristic and helps to differentiate from the quicker (sometimes simultaneous) involvement of several cerebral nerves in the course of an acute or a subacute encephalitis. Moreover, in encephalitis, fever and other signs of infection usually precede the occurrence of the symptoms that suggest brain tumor.

DIFFERENTIATION OF EPIDEMIC ENCEPHALITIS FROM CERTAIN OTHER NEUROLOGIC AND PSYCHIATRIC SYNDROMES

Myoclonias.—During the past two years myoclonic forms of epidemic encephalitis have been frequently observed. Excellent descriptions have been given by Sicard and Kudelski (1920) and by Boveri. When encephalitis is epidemic, the occurrence of an acutely developing myoclonia will cause little difficulty in diagnosis, but when there is no epidemic a sporadic case of myoclonia would scarcely be suspected to be encephalitis, or if an encephalitic origin were thought of, differentiation from other myoclonic states (for example, progressive paramyoclonus multiplex) might not be easy. Myoclonia of the abdominal muscles, first unilateral, then bilateral, is a valuable encephalitic sign (T. F. Reilly). Myoclonic contractions of the diaphragm may simulate ordinary hiccup, and it is interesting that epidemic hiccup has been reported (Gotti in Italy; Boyd in Manitoba). A myoclonia that is latent can sometimes be induced by placing the limbs in certain positions or by tapping the tendons (P. Sainton).

An interesting example of hemiclonia alternans has recently been reported by Sicard and Kudelski. There was rhythmic clonus of the right arm and of the left side of the face with paralysis of the left nervus oculomotorius.

Chorea.—Many choreiform manifestations have been seen since the hyperkinetic forms of epidemic encephalitis have been prevalent. In Vienna, Dimitz described this form of the disease as "encephalitis choreiformis." Farquhar Buzzard of London (1919) has reported rhythmic movements of choreatic or of athetoid type that set in weeks or months after the attack of encephalitis. In Baltimore, Happ and Blackfan have described choreiform states in association with pathologic behavior and persistent insomnia in young children suffering from encephalitis. In France, Lesné and Langle, Souques and Harvier and Levaditi have reported certain cases of acute febrile chorea that they believe is a manifestation of epidemic encephalitis. Both chorea minor and chorea major have been simulated by encephalitis. Syndromes like those designated as Dubini's disease, Henoch's electric chorea of young children and Bergeron's electric chorea of older children may all be produced by epidemic encephalitis. When encephalitis is epidemic, therefore, its existence must be thought of when, in association with signs of infection, choreatic disturbances of motility are encountered. A careful analysis of each case may help to reveal the primary disease of which the chorea is a manifestation.

Parkinson's Disease.—Parkinson-like syndromes, especially imitating paralysis agitans sine agitatione, have been frequently seen among the encephalitic cases in all countries. The acute development of this amyostatic syndrome (rigidity, characteristic attitudes, facial mask and poverty of movement with or without tremor) has been a striking feature of this type of encephalitis. In some of these cases the cell count in the cerebrospinal fluid has been increased and globulin has been present, but in other cases the cerebrospinal fluid has been entirely negative.

This Parkinson-like picture is, doubtless, like other parkinsonian syndromes, dependent on a disturbance of the extrapyramidal motor innervations due, in all probability, to lesions involving the nucleus lentiformis of the diencephalon, especially the globus pallidus (J. Ramsay Hunt). P. Marie (1920) maintains that the parkinsonian syndromes of encephalitis differ from genuine paralysis agitans, the latter being rare before 40, unassociated with fever, insidious and slow in onset with steady progression, most often accompanied by a characteristic tremor, not accompanied by disturbances of the tongue or of opening the mouth.

Cataleptic or Catatonic States.—In the first English epidemic, many of the cases were described under the name "epidemic stupor" and in other countries cataleptic states, catatonic states and waxy flexibility have been described as part of the symptomatology of many cases. One of my patients, a physician, when first seen, lay like an image, devoid of spontaneous movement; he was unable to swallow; he answered questions with great difficulty and only after a long interval; he was unable to read (probably on account of accommodation paralysis) and had retention of urine and slight leukocytosis. There was general muscular rigidity, the agonists being involved equally with the antagonists, but there was no evidence of spasticity, no Babinski sign. The face was expressionless. The condition gradually passed off, and this man is making a good recovery.

In asylums for the insane especially, the differentiation of this amyostatic syndrome from the catatonic states of dementia praecox and the cataleptic states of hysteria may sometimes be difficult, but a consideration of the mode of onset and of the course will usually clear up the diagnosis, especially when encephalitis is epidemic. E. D. Bond (1920) emphasizes the fact that mild and transient though definite symptoms of epidemic encephalitis may easily be overlooked in hospitals for mental disease when these symptoms occur in patients who are excited, secretive or indifferent.

Myasthenia Gravis.—Several of the patients suffering from encephalitis in this epidemic have at first been thought to be suffering from myasthenia gravis, owing to certain manifestations (profound asthenia, general muscular weakness, and especially weakness of muscles innervated by the motor cerebral nerves, exhibited as ptosis, facial paralysis, dysmasesia, dysarthria and dysphagia). But myasthenia gravis is a rare disease. It is afebrile. Its course is marked, characteristically, by remissions and exacerbations. On electrical examination, the myasthenic reaction of Jolly is obtainable. There is no atrophy and no reaction of degeneration of the paralyzed muscles.

Progressive Central Muscular Atrophy.—Either the spinal or the bulbar form of progressive (central) muscular atrophy may sometimes be simulated by the more subacute forms of epidemic encephalitis. But it is rare in progressive central muscular atrophy to see an onset as acute as is the slowest onset in epidemic encephalitis. Moreover, in true progressive (central) muscular atrophy there is no retrogression of the symptoms, even though there may be temporary arrests, whereas in epidemic encephalitis, if the disease is not soon fatal, recovery may be complete or improvement be marked, some of the atrophied muscles regaining their normal volume and the fascicular and fibrillary twitchings disappearing.

Hysteria and Neurasthenia.—Some of the more obscure forms of epidemic encephalitis have been erroneously supposed to be cases of hysteria or of neurasthenia. The pathologic drowsiness of encephalitis may be confused with hysterical twilight states or hysterical narcolepsy. The residual neuralgias, the restlessness, the insomnia and the disturbed mentality of the convalescent encephalitic may be ascribed to neurasthenia or to psychasthenia. In encephalitis it is exceedingly uncommon not to be able to demonstrate at least some signs of organic disease (pupillary disturbances; partial paralyses of cerebral nerves, alterations in the cerebrospinal fluid, etc.).

CONCLUSIONS

1. On account of the toxic component and of the inflammatory infiltrative component of the disease process, both general and widely disseminated focal manifestations of involvement of the central and peripheral nervous systems may occur in encephalitis and give rise to the most diverse disturbances of motility, of sensation, of coordination, of the reflexes and of the psyche.

2. Despite the enormous number of clinical forms encountered in epidemic encephalitis, there is a marked tendency to the repetition of certain characteristic forms or types, of which the somnolent-ophthalmoplegic, the paralytic, the amyostatic and the hyperkinetic are the commonest.

3. When the disease is epidemic, it can usually, in outspoken cases at least, despite the clinical diversity, be easily recognized, though in abortive, imperfect, rudimentary and aberrant cases great difficulties in diagnosis may be experienced, and doubtless many cases remain entirely unrecognized.

4. The occurrence in a patient of (*a*) pathologic drowsiness (lethargy), (*b*) cerebral nerve paralysis (especially ophthalmoplegia), (*c*) an acutely developing parkinsonian syndrome, (*d*) a cataleptic or a catatonic state, (*e*) myoclonia, (*f*) chorea, (*g*) pupillary disturbances, (*h*) violent neuralgia, (*i*) a poliomyelitic syndrome, (*j*) a peculiar delirium, (*k*) a psychotic state or (*l*) signs of meningeal irritation in times when encephalitis is epidemic, should make one think of the possible existence of the disease.

5. Though epidemic encephalitis may simulate any one of a large number of neurologic and psychiatric syndromes of entirely different origin, the mode of onset, the course, and the results of carefully conducted neurologic and psychiatric examinations (including an examination of the cerebrospinal fluid), will usually yield the diagnostic criteria that suffice for its recognition and differentiation.

PHYSICAL FINDINGS IN THE PSYCHONEUROSES*

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At the present time there are almost as many opinions about the etiology of the psychoneuroses as there are men who think about them. One end of the scale is sounded by those who believe that the neuroses spring entirely from mental repressions and conflicts. The antithesis of this is the absolutely organic theory which holds that psychoneurotic states are produced solely by physical disease. For some time it has been apparent that there is a tendency to depart from these two radical conceptions. Experimental studies and clinical observations show, beyond a reasonable doubt, intimate association between emotions and mental life in general and the physical state. We may still be honestly uncertain as to where to place the starting point of the neurosis: in the body, or in what is popularly known as the mind.

During the two year period, 1918-1920, 260 patients presenting the symptomatic picture of hysteria, neurasthenia, psychasthenia, anxiety states, etc., were examined and treated at the mental and nervous clinic of the Pennsylvania Hospital. Of this number, 140 revealed only minor pathologic changes. In the remaining 120, or 46.1 per cent., there was definite, and to my mind significant, organic disease. Given in the order of their frequency, we found: endocrine dysfunction, tuberculosis (including the pulmonic, intestinal and glandular varieties), syphilis, extensive apical abscesses, organic heart disease usually with beginning decompensation, postinfluenzal states, arteriosclerosis, anemia, combined heart and kidney disease, osteo-arthritis, sinusitis, infected tonsils, extreme visceroptosis, infected pelvic structures, chronic Neisserian infection, nephritis, chronic appendicitis, suppurative otitis media, prostatitis, gastric and duodenal ulcer, beginning gastric carcinoma, lead poisoning, floating kidney and diabetes. There were nineteen instances of ductless gland disturbance involving the thyroid, pituitary, and suprarenal glands; eighteen cases of tuberculosis, and fifteen patients with syphilis. Of the entire 120 cases, eighty-five were symptomatically expressed as neurasthenia, twenty as psychasthenia, twelve as hysteria and three as anxiety neuroses.

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* Read at a Clinical Conference of the Bloomingdale Hospital, White Plains, N.Y., March 30, 1921.

REPORT OF CASES

It may be worth while to summarize a few illustrative case histories.

CASE 1.—A widow, aged 37, with four children, had a combined hysterical and neurasthenic reaction. She complained of intense headache, both frontal and occipital and severe lumbar pain. There were numerous paresthesias. On the external aspect of the left thigh was a large, irregular area of sensory dulness. When this was touched by the bed coverings at night, marked discomfort and insomnia resulted. There were frequent "hysterical-like" paroxysms of weeping, and the patient constantly dwelt on her belief that she was becoming insane. The illness was of one year's duration. Previous to that time she had operated a 5-pound hammer at a steel mill. She had been under medical treatment for eleven months, and the diagnosis was neurasthenia.

The blood pressure was 215 mm. of mercury. The ophthalmologist reported marked sclerosis of the eye vessels. Further study and subsequent improvement under treatment made it evident that the neurosis was largely conditioned by organic changes, of which the unusually high blood pressure was one important manifestation.

CASE 2.—A widow, 28 years old, with four young children, was "nervous," worried, cried frequently and constantly felt a lump in her throat. There were insomnia, headache, "sparks" before the eyes, a numb sensation in the hands, and a feeling in the head as if two close-fitting wheels were "grinding together."

She had a neurotic make-up, and she had had several terrifying experiences. At the age of 12, she discovered her father hanging from the staircase—a suicide. Shortly before this she had witnessed the death of an older sister by drowning. Some years later there was a fire in the factory where she was employed, and the patient became rigid and unconscious. Her husband died of tuberculosis about a year before.

Examination revealed inactive tuberculosis, beginning hyperthyroidism, a badly lacerated perineum, uterus with inflammatory changes, advanced pyorrhea with many apical abscesses, and chronic otitis media with offensive discharge.

The course of treatment was first complete rest in a convalescent home, then the teeth roots were extracted, the ear condition cleared up and the torn pelvic structures repaired. Hand in hand with these reconstructive measures, there were psychotherapeutic treatments. She is now practically well.

CASE 3.—A woman of 30 with an infant was described as a "nervous crank" who "screamed whenever anything went wrong" and "had a mania for going to hospitals and dispensaries." Her reaction was essentially neurasthenic. Fatigue, aches, pains, discomforts, and odd sensations were the burden of her story. There were incipient pulmonary tuberculosis, systolic blood pressure 90, diastolic 52, muscular adynamia, bronzing of the skin and thyroid struma. The progress of the condition confirmed the diagnosis of Addison's disease.

CASE 4.—A woman of 39 presented a pronounced anxiety state with phobias, the most prominent being a fear of dire calamity if she should leave her house except on the most urgent errands. The neurosis had followed the death of her husband a year before. He had been extremely jealous and would leave his work two or three times a day to find out whether his wife was at home. Examination revealed pulmonary tuberculosis, apical abscesses and a femoral hernia.

It is true that this patient presented important psychologic difficulties which needed to be adjusted, but psychotherapy alone would have left an unfinished problem.

CASE 5.—A man, 38 years old, was exceedingly timid, and his collection of phobias was increasing by leaps and bounds. He was "nervous" and "shaky," could not eat and feared that he was losing his memory. The patient was a painter. He often had gastro-enteritis with severe abdominal pain and had lost about 30 pounds in weight. There was advanced pyorrhea, and the roentgenogram revealed extensive apical abscesses.

Here, there was apparently no need for psychotherapy. Rest, change of occupation and attention to the teeth have resulted in a speedy recovery.

CASE 6.—A woman of 60 presented a neurosis which came on with "a noise in the ears" following her daughter's death. The tinnitus kept her from sleeping. Soon a number of phobias developed, chiefly the fear of being alone and the fear of going to bed. She began to sit up all night. There were constant drawing pains in the head and arms. The otologist reported severe catarrhal otitis media with partial deafness. The roentgenologist found apical abscesses, the laboratory a low grade nephritis, and the internist a visceroptosis. Under treatment this patient is showing marked improvement.

COMMENT

How are we going to regard the organic morbidity of the psychoneuroses? Perhaps, after all, the occurrence of tuberculosis, syphilis, ductless gland disturbance, heart and kidney disease, apical abscesses, etc., is a matter of coincidence, and no relationship at all exists between these diseases and the neurosis. This would seem more plausible if these factors were unusual and infrequent, but when they may be discovered in almost 50 per cent. of our patients, it seems likely that we are dealing with something more than mere chance association.

Students of heredity will be apt to regard this physical inferiority as an expression of so-called psychopathic inheritance. It is, of course, true that this might influence the particular variety of mental reaction. However, if it were solely a question of physical inferiority, one would expect it to take a more uniform type; and further, to assume a more typical clinical syndrome. In our patients twenty-six more or less common diseases were represented, conditions encountered every day in general practice.

I have heard the objection that these cases merely simulate the neuroses and that basically and essentially they are not hysteria, neurasthenia, psychasthenia and anxiety states. If they are not, they certainly are often faithful duplicates. After all, a diagnosis must be made on the symptoms, and the picture of neurasthenia, for instance, is too clear to escape recognition.

May we assume a direct causal relationship? If so, how does the organic disease operate? Dana [1] deplores the emphasis placed on the psyche as the sole etiologic factor in the psychoneuroses. His argument

1. Dana, Charles L.: The Somatic Causes of Psychoneuroses, J. A. M. A. **74**:1139 (April 24) 1920.

is based on the theory of "synapsis" which implies "breakable points and points of variable resistance in the finer organizations of the brain." Here the traveling nerve impulse meets its highest resistance, and neural phenomena are apt to develop. Paths of conduction may be blocked or made free not only by mental, but also by physical agencies and endocrine disorders, toxemias of various kinds, etc., may all act as etiologic agents. Wechsler [2] found marked signs of dysfunction of the pituitary gland in four typical psychoneurotic patients, of the thyroid gland in three, and of the suprarenal and sex glands in two. Treatment was successful, and the author seeks in "disturbance of the glands of internal secretions a source of rational therapy." Robertson [3] is convinced that chronic bacterial infections are an important element in the etiology of neurasthenia. The nasal passages, the lower respiratory tract, the nasopharynx, the mouth, the fauces, the intestinal and genitourinary systems were investigated in sixty-six cases. The *B. influenzae*, the pneumococcus, several strains of streptococci and diphtheria bacilli were isolated and cultivated. Immunization produced important therapeutic results. Carver and Dinsley [4] made some interesting observations on healthy soldiers in an "ammunition proof and demolition section" during the course of their ordinary duties. They were well sheltered, and the element of fear was minimal. Various symptoms were noted, including active tremors, increased pulse and respiration rates, and in a few cases, vomiting. Those most affected became less resistant to subsequent exposure. As a result of their experiments and of observations on soldiers, the authors believe that the "indirect concussion" of high explosives produces definite changes in the central nervous system accounting for certain of the symptoms usually ascribed to psychogenic factors.

Obviously it is unfair and dangerous to dismiss the psychoneurotic without adequate examination. Consultations, serologic, biochemical and radiographic studies may all be needed for our difficult problems. Finally, it seems evident that no single system is sufficient for the treatment of the neuroses. When we really do not know it is not safe to assume or to theorize, especially if our hypotheses mean that etiologic possibilities are to be excluded in actual practice.

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2. Wechsler, F. S.: The Psychoneuroses and Internal Secretions, Neurol. Bull. **2**:199 (May) 1919.

3. Robertson, W. Ford: The Infective Factors in Some Types of Neurasthenia, J. Ment. Sc. **75**:16 (Jan.) 1919.

4. Carver, Alfred, and Dinsley, A.: Some Biological Effects Due to High Explosives, Brain **42**:113, Part 2, 1919.

News and Comment

DUTCH SOCIETY OF MENTAL HYGIENE

Headed by Dr. Ariëns Kappers and Dr. B. Brouwer, a society for the scientific study and promotion of nervous and mental therapeutics has been formed in Holland. It includes all the professors in neuropathology and psychiatry in Holland and several pharmacologists. The society is sustained by subscriptions. It publishes a periodical called *Neurotherapeutics*, for which contributions are accepted in all modern languages. Contributors are paid $13 for each sixteen pages of printed matter. The society also invites foreign physicians to deliver lectures on their work, Professor Petrén of Sweden and Prof. F. Plaut of Munich being among recent lecturers. It is expected that Levaditi will lecture to the society in October. In an announcement, physicians are offered membership for the annual fee of 10 florins, life membership for 250 florins or associate membership for 5 florins yearly or a single sum of 100 florins. The society may be addressed through the secretary, Dr. B. Brouwer, Koninginneweg 170, Amsterdam.

FIRST SESSION OF THE FRENCH LEAGUE OF MENTAL HYGIENE

The first session of the French League of Mental Prophylaxis and Hygiene was held in Paris, June 1. Addresses were delivered by the president, Dr. Toulouse, and by Justin Godart who defended the right of the insane to support by the government, by Prof. Jean Lépine who requested the creation of dispensaries for psychopathic patients, and by Dr. F. Williams, director in France of the Rockefeller Foundation. Requests for admittance to membership in this organization should be addressed to the secretary, Dr. Genil-Perrin, Avenue de la Bourdonnais 99, Paris, 7th arrt.

Abstracts from Current Literature

APHASIA: A HISTORICAL REVIEW (HUGHLINGS JACKSON LECTURE FOR 1920). HENRY HEAD, Discussion by Drs. James Collier, James Purves Stewart, Kinnier Wilson and others, Brain **43**:391, 1920.

The Hughlings-Jackson lecture for 1920 by Head is a very complete and interesting historical critical review of the subject of aphasia. The story of the influence of Joseph Gall, Broca, Bouillaud, Trousseau, Jackson, Bastian, Baginsky, Wernicke, Lichtheim, Moutier, Déjerine and others on the conceptions which we have held in the past concerning this subject is interestingly put forth.

Gall, who localized speech in "those portions of the brain which lay in relation with the posterior part of the orbital cavity," . . . "for as a boy he noticed that his companions who had prominent eyes had a gift for languages and a memory for words," is given credit for being the first to take into account the fact that speech and intelligence are probably cortical functions. Mention is made of Broca's studies and of the fact that the brain exhibited by Broca at the Societe d'Antropologie, which served as a basis for Broca's idea of the localization of the speech function, was not sectioned and properly examined anatomically until Pierre Marie did this forty-five years later. The fact that Marie was able to disprove Broca's deductions by his studies of Broca's material is also mentioned. A careful review is made of Jackson's views, who, for the first time, definitely divided healthy language into the (1) intellectual, i. e., the power to convey propositions, and (2) emotional, i. e., the ability to exhibit states of feeling. Jackson believed that these two parts of speech were separated by disease and that in most cases of disease the former only was affected. As Hughlings Jackson said, the question is not how is general mind damaged, but what aspect of mind is damaged in aphasia?

Head treats Wernicke's views, particularly in his discussion of the case of isolated agraphia, with scant respect, and likewise shows little patience with the views and diagrams of Lichtheim and of many others.

Marie referred the aphemia (motor aphasia) of Broca to difficulties in the lenticular zone, while the aphasic side of speech defects (sensory aphasia, auditory aphasia and verbal amnesia) were referred to disturbances in Wernicke's zone. According to Marie, the true aphasias are a disturbance of general intelligence and of special intelligence for language. Head gives some attention also to the controversy which took place between Déjerine and Marie over the latter's views. He speaks of these debates, which at times became very bitter, as purely verbal battles, which would have resulted in some clarification of their points of view had the disputants been familiar with Hughlings Jackson's ideas. Into these Head goes rather minutely and points out again that Jackson was a pioneer in classifying aphasics properly into two great classes; first, those who are speechless or practically so, and second, those who have a plentiful vocabulary but who use words in a wrong way. He agrees with Jackson that there cannot possibly be such a condition as pure agraphia because a man who cannot write spontaneously may be able to copy printed matter in perfect handwriting. "He cannot write voluntarily because

he has lost the use of written words in propositions." The fact that such patients can write their names and addresses or those of intimate friends or relatives is ascribed by Jackson and Head to the theory that these things "with time more nearly reached the level of an automatic act." Head emphatically agrees with Jackson that in the majority of cases of aphasia mental images are not affected. As Head says "It is not sufficient to hold up some object and ask the patient to name it . . . his power of responding must be tested by a series of observations in which the same task occurs on two or more occasions and each of the tests must be put before the patient in several different ways . . . so that the choice of any one object (in the tests) recurs three or four times in the course of the series." Head gives a short abstract of a description of his tests which have been described in *Brain*, volume 43, pages 91 to 107. Then follows a summary of Head's views which were also expressed fully in that paper and were abstracted by the reviewer in the ARCHIVES OF NEUROLOGY AND PSYCHIATRY, December, 1920.

Head's ideas might be summarized in his statement that "we should as soon expect a special center for eating as for speech . . . no lesion, however local, can affect speech and speech only. Cerebral injury disturbs certain physiologic processes which subserve the complex acts which we speak of as speech. Any organic injury which produces a disorder of speech disturbs other functions not usually associated with language, or leaves unaffected much that undoubtedly belongs to speech." Thus Head disposes of all the old terms with their underlying conceptions, such as motor and sensory aphasia, apraxia, agraphia, or verbal amnesia. Head ends this summary of his views by saying, "No disorder of speech due to a unilateral lesion of the brain corresponds exclusively to any one of these categories; still less can these hypothetical conditions be associated with limited destruction of any one part of the brain . . . any mental process is liable to suffer which demands for its performance exact comprehension, voluntary recall and perfect expression of symbolic representations."

At this point the reviewer wishes to take the liberty of pointing out that several times in Head's recent writings the statement is made that intelligence as such is something entirely apart from speech and may be unaffected by disease processes causing aphasia.

If Head's views just quoted are true, how can it be possible that any aphasic will not show some disturbance of the intelligence? Before this point can be intelligently discussed every one must have clearly in mind what is meant by the word intelligence. Either one agrees with Marie that there is a special intelligence for speech and that no disturbance of speech resulting in aphasia can possibly occur without some disturbance of the intelligence, or he believes with Head that intelligence is developed only incidentally with speech and that it is possible to damage seriously the speech function without disturbing the intelligence, or he agrees with still others (see Purves Stewart's discussion) who hold tenaciously to the speech center theories and their diagrammatic conceptions. The latter would have us believe that the speech function is more or less sharply localized in various points of the brain and that it is possible to injure these areas with consequent disturbance of speech in which only that function and nothing else is disturbed.

Head's statements just quoted would seem to place him in the same position as Marie and the reviewer who believe that intelligence and speech develop apace and are intimately bound up together in the brain. This is so because the mind which thinks does so only in speech or, as Head calls it, symbolic

expression; whether that be spoken or not is immaterial. This on the anatomic side simply means that the entire brain is connected up in an intricate, complex way by means of short and long association paths and that many, if not all, of these pathways must be used in ordinary symbolic expression. It is, therefore, ridiculous to talk of speech centers. There must be in areas of the brain that are fairly well known, which necessarily vary in development and localization with persons, certain places where memories of various kinds are stored. These association areas are connected with each other and with various parts of the brain having other functions by certain pathways which, when interrupted, not only disturb the speech function but necessarily disturb also other functions of the brain which, taken all together, make up general intelligence. As we understand intelligence, it is a combination of all the memories and experiences of the person which are capable of being expressed in thought. As Head well says, "The higher the propositional value of the mental act, the greater difficulty will be present." General intelligence certainly is affected in those cases of Head's which he calls semantic aphasia. His idea is that in these cases there is an inability to recognize the significance of words and names. The reviewer believes that if a person is unable to do this certainly his intelligence generally must be seriously affected.

Dr. James Collier, in his discussion, said that the clinical varieties of aphasia described by Head "correspond to definite stages of dissolution or recovery: neither do they reveal directly the elements out of which language is built up. On the contrary, they show the components into which a highly complex state of psychical processes can be separated by destruction of certain portions of the brain." Collier further said that the "alterations in speech function which result from lesions of the brain are not in terms of the primary elements out of which speech has been evolved, nor do they reveal the elementary basis of the acts of speaking, reading and writing, but that they are in terms of the complete and highly developed function, which fails progressively from its highest to its lowest with increasing degrees of damage."

The reviewer believes this situation is similar to the tearing down of a house, for one can see in the débris the elements out of which the house was built. If the functions of speech and intelligence, which are localized in the cortex of the brain, are destroyed or disturbed by disease or injury, it is difficult to see why we are not justified in concluding that the resultant defects are important portions of the mosaic from which they have been removed, study of which may give important clues as to how intelligence and speech are patterned in the brain. If this is not true, then there is little importance in studying this subject, and the possibility of ever learning how speech and intelligence are developed must be given up as hopeless.

Head then goes on to review his conceptions of the four types of aphasia as classified by him, namely, verbal, syntactical, nominal and semantic aphasia. He, of course, admits that none of these groups is clear cut and that many of the cases merged some of the characteristics of each group; he offers them simply as a means of study and convenient grouping. As a matter of fact, the semantic and nominal groups seem to show a great many points of similarity.

Collier's discussion is chiefly important for the several points on which he differs from Head. He believes in the "existence of afferent paths to the speech region on the auditory side and on the visual side and efferent paths therefrom, by which speech is usually exteriorized, both spoken and written, and in these paths there is surely strict anatomical and physiological separation."

Collier believes that an "isolated involvement is possible if a lesion hits one of these anatomical tracts before it merges with others or breaks up into associated pathways." He disagrees with Head and agrees with Déjerine that a case of pure alexia may occur, and he claims to have seen the lesion demonstrated in the posterior limit of the speech area. While Collier agrees with Head that pure word deafness does not exist, he thinks that pure agraphia and pure motor aphasia may occur. Yet Collier says "there are cases which all of us must have seen in which lack of acceptance of spoken speech stands out in undue contrast with the acceptance of visual speech." He lays great stress on the recognition of speech "by means of the auditory path which allows of analysis of the correctness and fitness of what we have said and of its correction, if faulty." Collier also emphasizes the importance of the feel of speech, i. e., the sensations which accompany the movements of the speech mechanism. He says that when both these things, but especially the auditory immediate consciousness of the correct execution of speech, fail, then confusion must certainly be the result in thought and speech. Collier believes that the visual and auditory speech areas and functions cannot be sharply separated because the "function of visual speech, acquired at a much later age than is auditory speech, tacked on as it were to an already perfect and deeply impressed function of auditory speech, learnt by means of an already well versed auditory speech function, must occupy the same physiologic substratum as does the auditory speech function and is separately localized only in so far as its chief afferent and executive paths are concerned."

Collier agrees with the principle expounded by Head that the alterations in speech function, which result from lesions of the brain, do not reveal the elementary basis of the acts of speaking.

The reviewer has already discussed this point and in further disagreement would point out the fact that a good deal of the physiology, for instance, of automatic associated movements and tremors, and practically all of the knowledge that we have concerning these conditions has been taught us by the study of the pathology of these functions in the basal ganglions and in the midbrain. Why this cannot be so with speech is difficult to understand. Collier agrees with Head that the cerebral cortex concerned with speech functions acts as a whole and not in terms of isolated localized functions. He disagrees with Head's conception of syntactical aphasia and does not believe that this condition can arise from a lesion of the cortex. He thinks that jargon aphasia is a thing quite of itself and is characterized by overaction and volubility. In this condition the intelligence and quickness of apprehension stand out prominently, but the volubility seems to be due to a severance from control and lack of cortical guidance. Collier thinks that this condition is due to a blocking of the auditory afferents. He speaks of two patients who pathologically showed subcortical tumor "undercutting the posterior and mesial aspects of the temporal lobe, comparatively far from the cortex which was in no way involved." Collier says, further, that jargon aphasia is hardly ever encountered in the common lesions of the temporal region . . . "where the temporal cortex is likely to be involved." The reviewer believes also that jargon aphasia is due to a progressive lesion. An extension of the lesion to the temporal cortex causes the jargon aphasia to lessen in volubility and finally to disappear.

The discussion by Sir James Purves Stewart can be summed up in a few words. He sees no reason to change the conception of psychomotor aphasia and psychosensory aphasia and proceeds to discuss six cases of aphasia result-

ing from craniocerebral wounds (with one exception which was a vascular lesion in a man aged 55). Stewart's cases are not completely given. There is no record of a complete neurologic and aphasic status and nothing is said about the handedness of the patients or of their previous condition and education.

It occurred to the reviewer in reading Stewart's cases and even those which were the basis for Head's studies to ask what tests the observers had made to determine that the injuries were purely limited to one side of the brain. How could they know that there was not more than one lesion on both sides of the brain? Nothing in the reports of Head's or Stewart's cases answers this question. There is a lack of complete neurologic status and certainly a complete lack of pathologic report with which to exclude this important point. Stewart believes that the old conception of psychovisual, psycho-auditory and psychomotor speech centers are justified.

The discussion of Kinnier Wilson follows and seems to be in perfect agreement with our point of view concerning the type of case used for both Dr. Head's and Dr. Stewart's studies. Wilson points out that even Head admits that gunshot wounds "tend to cause greatest damage on the surface of the brain in addition to which there is the factor of commotio in all such patients. The fact that small hemorrhages may catch projection fibers where they converge or diverge is, in my opinion, much less important for the subject of aphasia than that association, i. e., trans-cortical fiber systems should be involved . . . the ideal case is neither the tumor nor the gunshot wound case but that with isolated vascular lesion, the exact position of which there is opportunity to establish postmortem."

Wilson takes exception to Head's interpretation of the difficulties shown by patients with aphasia in imitating movements when confronting the examiner as against the ease with which the same movements are performed when the patient sees them in a mirror, the examiner being in back of him. Wilson does not believe that these difficulties occur because of disturbed verbalization but are due to a disturbance of automatism resulting in dyspraxia or apraxia. It seems to us, however, that Head's interpretation of the difficulty is the true one because, in attempting to imitate movements not commonly used, a certain amount of thought in the form of internal speech is necessary so that one may attend to the matter in hand. Of course, Wilson's interests are along the lines of establishing in apraxia the same sort of diagrammatic fallacies that we have been so long in breaking down in aphasia, and his entire discussion is directed along these lines. He quarrels with Head's grouping of the cases and does not see why it should be necessary to discard our old terms with some of their underlying conceptions. Particularly does he disagree with Head's fourth classification (semantic aphasia). He believes that these cases are really nothing but the agnosias described by Liepmann and others.

The reviewer has on a number of occasions called attention to the fact that the questions of aphasia, apraxia and the intelligence are intimately bound up, one with the other, and any attempt to separate them definitely as functions or "faculties" of the brain will result in artificial classifications and conceptions which run counter to established physiologic, psychologic and anatomic facts. Saying, as Wilson does, that motor aphasia is nothing but an apraxia as applied to speech is a truism. This, however, has not helped to explain to any great extent, in the absence of any sensory or motor paralysis of the organs involved in speech or motion, the mechanism either of disturbances of speech (aphasia) or of volitional movement (apraxia). So, too,

to call the sensory aphasias a form of visual or auditory agnosia does not help matters.

Dr. Stanley Barnes in his discussion obviously has failed to read Head's original article carefully for he expresses a desire "to have heard more emphasized by Dr. Head that intellectual faculties were so closely bound up with speech that one must expect them to be represented in the same area of the brain and that any serious defect in speech must mean a serious defect in intellect too." Head has definitely said that the opposite of this statement is true. Barnes, however, believes that "the two functions, speech and intellect, were largely interdependent and were represented in the same area of the brain."

Dr. Barnes' discussion agrees so closely with what the reviewer thinks of this whole subject that to dilate on it would simply be to repeat this statement over and over again in different words.

The psychologic discussion by Dr. James Herbert Parsons sems to be the basis for the same sort of a conception as that expressed so broadly by Dr. Barnes and in a more narrow sense by Head. Parsons sketches rather interestingly the psychology of the development of "meaning" and shows how intricate a process learning is and how impossible it must be to have the function of speech and thought localized in any circumscribed areas of the brain. The process is one of delicate and intricate association, and our thought and speech are learned in a variety of ways through numerous efferent sensory pathways with boundless primary and secondary associations resulting in a complicated function.

Head in reply could not understand why Dr. Collier wished to keep up terms used in describing aphasia which were physiologically, anatomically and psychologically impossible. For these reasons Head had sought to replace them by something more fundamentally correct.

In describing syntactical aphasia Head says that the important thing is that such patients lose the regulating power over the rhythm and balance of what is said because speech is not recognized in a critical way by the patient. The words spoken by persons with syntactical aphasia are expressions of the speech functions which are not disturbed. Head disagreed with Collier strongly in his conceptions of visual speech or auditory speech; he says that when a person speaks "his actions are carried out by psychological processes above the visual and auditory levels." Head explains that his object in selecting the cases studied by him was to see the actual forms assumed by destruction of speech regardless of the nature of the lesion, whether it was cortical or subcortical . . . "it was of no use trying to localize the position of an unknown function on the surface of the brain." He strongly objects to the use of the term "faculty" as applied to speech and intelligence and says that such conceptions come down from medieval philosophy. He is particularly strong in his objections to the rather old and conservative stand taken by Dr. Stewart.

What Head thinks about Wilson's discussion is summed up in his statement that "the moment one drove apraxia to definition one saw that it did not exist . . . but when one used the word apraxia to explain the loss of speech it did not seem to explain more than the most simple of aphasias. He wished to get rid of all such terms."

In answering Dr. Barnes, Head said that he did not believe in "localization . . . of so-called motor aphasia or sensory aphasia" because he did not believe in the existence of motor and sensory aphasia." Head agrees with Jackson that there is no such thing as general memory, and that psychologists nowadays did not believe in general intellectual defects. There are only

definite things remembered. There is no such thing as general memory. "Speech is a form of intellectual activity; therefore, in saying there was a defect of those processes which were responsible for speech, it was assumed there was a defect of intellect.' But this was a specific loss of function and there was not that gross widespread defect of intellectual aptitudes as was found in the slightest case of dementia."

To this point the reviewer has called attention previously. He is in accord with Head, the only difference being in the interpretation of what intellect and intelligence mean. If one does not use intelligence as a general term, meaning by it that he is intelligent who has attended to and stored correctly many memories and clearly conceives their importance and significance, then he may come into complete agreement with Head. For Head and many psychologists there is no such thing as general intelligence but merely special intelligences or memories of specific things. This the reviewer, from the medical clinical standpoint, thinks is mere splitting of hairs.

OSNATO, New York.

DEVELOPMENT OF THE REACTIONS AND PLANTAR REFLEXES OF A BABY BORN BEFORE TERM AND UNTIL TWO YEARS OF AGE. H. BERSOT, Schweiz. Arch. f. Neurol. u. Psychiat. **7**:213-231, 1920.

The researches of Flechsig, Soltmann, and others, relating to the development of the anatomic structures of the nervous system placed our knowledge of this far in advance of that concerning the development of its physiologic functions. Structurally the nervous system develops rapidly during the first years of life, progressively less rapidly up to the twentieth year, after which involution slowly commences. Concerning the physiologic development much less is known, particularly the development that occurs during the intra-uterine period.

In attempting to establish the relationship between the anatomic features of the central nervous system and its function, the conclusion was reached that one must naturally have preceded the other. Most writers state that full development of given parts of the organ precede the development of their function, while others believe that function may precede the full development. An indeterminable discussion has resulted, centered mainly around these facts: Up to the middle of the fifth month only gray matter is found in the fetal nervous system, after which myelinated fibers appear; on this fact the assumption was based that the first movements of the fetus are noted in the fifth month of intra-uterine life. In contradistinction to this, Preyer states locomotor movements are noted from the seventh week of embryonic development. A similar dispute has centered about the observation that after the sixth month following birth there is a rapid disappearance of the extension of the large toe on plantar irritation, which is coincident with the development of myelinization of the pyramidal tracts; that incomplete myelinization of the pyramidal tracts is the cause of the Babinski reflex.

What characterizes the responses to external excitation obtained in an infant is the fact that they are generalized. The organism as a whole responds. The baby cannot localize, cannot vary its reactions and does not differentiate. In this respect it differs fundamentally from the adult, in whom a particular excitation produces a localized response that varies as the excitation varies. Thus, in a baby, in tapping the back with a pin, motor reactions appear in both lower extremities and toes. In the adult, on similar excitation, one

obtains only a feeble local reaction, the lower members remaining immobile. If the excitation is more marked and painful, one obtains, in an adult, reactions which approach those in a baby following slight excitation. The reactions which are not produced in the adult on slight excitation are supposed to exist in a latent manner and are not completely abolished. Therefore, there is no fundamental difference in the reaction of a baby as compared with that of the adult; the difference is one of degree, variability and multiformity.

It is generally stated that during the first months of life the Babinski reflex turns out positive. C. Cattaneo found it present in 30 per cent. of cases during the first three months of life, then progressively less frequently up to the end of the second year. According to Muggio, after the sixth month of life, it is seen especially in rickets. According to Lowest-Morse, the normal plantar reflex is not seen until after the second year. Léri, from a study of 166 infants, concluded that at birth extension of the toes is the rule, flexion is the exception. After 3 years, the opposite is true. Between the ages of 1 and 3 extension of the toes is rather indicative of affections of the pyramidal tract. The pyramidal tract, having scarcely had its myelinization completed, is more sensitive to toxic degenerations than that of the adult and is readily affected in general nutritional disorders. According to him, extension of the toes normally disappears at about the fifth or sixth month. This disappearance is coincident with the disappearance of the general spasmodic attitude of the new-born.

S. Galant, in studying the plantar reflex of 132 infants, found the sign of Babinski present in ninety-eight cases and fanning of the toes in thirty-five; the latter is seen especially in babies up to the fourteenth day; after the second week fanning of the toes is rare. Galant noted that the older the child, the less frequent the Babinski phenomenon and the more restricted the reflexogenic zone. By repeated excitations of the sole, except in very young babies, one is able to suppress the Babinski sign and elicit the so-called normal plantar reflex.

The reflexes of a baby cannot be considered as fixed and immutable as those of the adult. It is this variability that Bersot is particularly interested in. In preceding studies of reflexes during the entire life, Bersot showed that the plantar response diminished in intensity until the twentieth or thirtieth year, at which time it reached its most feeble value, and following which it again became augmented. He showed that a plantar reflex was present during intra-uterine life, when it took the form of part of a general reaction, becoming more and more differentiated and localized after the third or fourth months following birth. Fortunately, Bersot was able to obtain a fetus to carry out his study.

According to Preyer, movements appear after the seventh week; these motor reactions have characteristic features. Soltmann states that the excitability of the fetus is less feeble; that the muscular movement possesses a certain slowness of reaction such as is seen in the adult on great fatigue or in a number of pathologic conditions. According to A. Westphal, fetal muscles are inexcitable by the electric current. Movements interpreted by Ahlfeld and Reubold as deglutition and by Mermann and Ahlfeld later as regurgitation are also seen. Ahlfeld also stated that rhythmic movements at the rate of 50 to 60 times per minute are visible to the attentive observer and correspond to movements of the thoracic wall of the infant. These have been traced by kymographic methods and have been interpreted as respiratory movements which are so slight in amplitude that the amniotic fluid does not enter the lungs. The fetus studied by Bersot was of four to five months', seventeen to

eighteen weeks' development; it behaved very much as in a state of sleep in which external excitation could scarcely arouse it. On irritation, feeble movements were noted in the extremities, the fingers and toes were immobile, the respiratory movements were slight, although the mouth was opened and there was no cry. As soon as stimulation was discontinued the infant at once seemed to plunge into a deep sleep.

Plantar excitation in this premature infant provoked only a flexion of the toes which was slow and feeble, but definitely present, and included all of the toes, particularly the outer four. The great toe frequently remained immobile. Of the four outer toes, the movement consisted principally in flexion of the first phalanges and extension of the two others; the toes seemed to lengthen and at the same time to go through a movement of slight plantar flexion, such as that produced by the combined action of the lumbricales and interosseus muscles. This movement was not accompanied by any other movement of the lower extremity or in the rest of the body and ceased after the sixth to eighth stimulation as though rapidly fatigued. This type of reaction was also noted by K. Crabbe. Oppenheim's reflex was not obtained.

At twenty-seven to twenty-eight weeks of intra-uterine life the infant presented the same reactions as those noted at birth. Irritation was accompanied by feeble crying of short duration. Plantar irritation here produced a retraction of the entire lower extremity by flexion of the thigh and leg which ceased as soon as the irritation was removed. The toes also went through a movement of flexion and elongation. The large toe remained either immobile or followed the movement of the other toes. There was also a contraction of the tensor fascia lata. On the opposite side the toes went through the same movement of extension with slight dorsal flexion of the foot.

At thirty-four to thirty-five weeks of fetal age the external appearance was similar to that of an infant born at term, except for a looseness of the skin due to lack of subcutaneous fat. The responses to irritation are more or less universal and are spasmodic or athetoid in character. On plantar irritation the four external toes undergo an extension, which is sometimes noted in the great toe also; however, more often this toe extends as in a positive Babinski reflex. The extremity is flexed at the ankle, the knee and the hip. Contractions of other muscles, such as the quadriceps and tensor fascia lata, are noted. The opposite extremity goes into a position of extension throughout. It is noteworthy that a baby born at thirty-five weeks has not the same reflex activity as that thirty-five weeks of age, but born at the thirtieth week. In the latter the reactions are more extended, more numerous and less feeble, as though reflexitivity developed by external irritation.

The reflexes of an infant born at term and up to six months of age are very much like those just described. The motor reactions are a little more lively, stronger and extended. Excitation of the foot usually produces a flexion of the toes rather than a positive Babinski reflex; however, extension of the large toe appears in the contralateral extremity before it appears in the homolateral one. At two or three months plantar excitation no longer produces flexion of the great toe, but a strong extension of the large toe and a movement of dorsiflexion of the foot. In the other toes there is an intra-osseus and lumbricales reaction. Contralaterally, extension of the great toe occurs with force and slowly and often remains at hyperextension during the entire observation of twenty excitations, which it is the custom of the writer to produce. There is less tendency to fatigue, and the feet tend to retain their position of defense for some time after the excitation has ceased. The general reaction, however, is marked.

At five or six months of age the reactions on the whole are the same as at the third month. These reactions constitute the plantar reflex. There is absolutely no way of distinguishing them as they occur normally from those noted in pathologic conditions at the same age. Bersot agrees with most writers that the Babinski phenomenon is present in the majority of cases. An important point, he thinks, is the generally increased reflexitivity of the infant. The entire cutaneous covering is reflexogenic, particularly up to the second or third month.

After three or four months the reflexogenic zones commence to become more limited. The plantar reflex can be elicited only by excitation of the plantar surface of the foot. In infants of six months the reactions are analogous to those of three months.

At nine to twelve months a distinction arises in that the infant seems able to discern different types of excitation. It does not react in a uniform manner to all external excitations. It remains quiet when it is uncovered, movements become better coordinated, and a life of relation seems to establish itself. Plantar irritation leads to retraction of the excited member with extension of the contralateral one. The toes usually go through a movement of plantar flexion, in which the large toe participates. There may be a flexion of both phalanges or flexion of the first with extension of the others. Extension of the contralateral great toe has completely disappeared by the age of nine to twelve months. At times it appears on the homolateral side, but more often has become replaced by flexion. The reflexogenic zone has become very much restricted.

At eighteen months, extension of the great toe is rarely observed. The general reaction of the organism is much diminished, muscular contractions are more rapid, movements quicker and more forcible, and the reactions themselves diminished in number, intensity and duration. Bersot's study led him to believe that the reaction in infants in particular must include all of the reflex movements rather than a limited observation of the particular member irritated. The article will be continued.

WOLTMAN, Rochester, Minn.

CONCERNING THE DIAGNOSIS AND TREATMENT OF HYPOTHYROIDISM. N. W. JANNEY and M. E. HENDERSON, Arch. Int. Med. **26**:297 (Sept.) 1920.

The authors emphasize the fact that early cases of hypothyroidism are frequently overlooked, although frank cases are easy to recognize. They report a series of cases in which, besides the ordinary history and physical examination, differential blood counts, glucose tolerance tests and basal metabolism determinations were made. The technic for the glucose tolerance test consisted in giving 1.5 gm. glucose per kilogram of body weight in 3 c.c. of water per gram of sugar and samples of blood taken immediately before the ingestion and at hourly intervals for two or more hours thereafter. Basal metabolism determinations were made with the Benedict portable apparatus.

Seventeen cases are included in the report, one of which was a definite cachexia strumipriva, and four were classed as dysthyroidism. As a group, the most important and frequent findings are: history of obesity, particularly in early life; history of glandular disturbances in the family; mental symptoms embracing dulness, irritability, headache, melancholia, poor memory and exhaustion; anomalies of development and liability to infections; physical findings which embraced hair anomalies, dry, harsh skin, pigmentation, atrophy,

frail, poorly developed nails, cold extremities and skin, stubbiness of hands and feet, dull expression, heavy lips, large tongue, saddlenose, increased panniculus, fat pads on the dorsum of hands and feet, in the supraclavicular fossae and on the back; subnormal temperature, pulse and respiration. The thyroid gland varied in size from normal to enlarged or decreased. The blood picture usually showed a mononucleosis. The average percentage in thirteen cases was: polymorphonuclears 55.1 per cent., lymphocytes 36.1 per cent. and large mononuclears 3.53 per cent., eosinophils being present in only one case.

Basal metabolism determination is pointed out as being the best means of establishing direct evidence of thyroidal disturbance, but it cannot be used as a sole and absolute criterion. The authors state that the glucose tolerance estimation showed that a hypoglycemia and a lowered blood sugar curve existed in hypothyroidism much more commonly than in hyperthyroidal conditions. In relation to the blood sugar curve it is pointed out that normally there is a return to normal in one and one-half to two hours, but in hypothyroidism it is considerably delayed and indicates a faulty sugar metabolism in the system. It must be remembered, however, that the same picture is sometimes given in disorders of the endocrine glands other than the thyroid. The basal metabolism rate did not follow the sugar curve in any sense, and it is concluded that the sugar curve is more reliable as diagnostic evidence earlier in the disease than basal metabolism ratings. The therapeutic test for deficiency of thyroid secretion is mentioned as filling a proper place in the diagnostic armamentarium and not to be forgotten.

Treatment is best controlled by basal metabolism studies since it indicates overfeeding or underfeeding earlier than the clinical evidence, although it must not be forgotten that in older persons it may be misleading, as the ability to respond is much slower clinically than in younger people, and a basal metabolism rate may be read as normal while the patient shows no change. So far as preparations of the thyroid gland are concerned in therapy, the authors emphasize the importance, scientifically and physiologically, of thyroxin which produces a much better response than any other preparation.

In regard to nitrogen metabolism in a person with hypothyroidism, one of the authors has shown by experimental work (reported elsewhere) that there is difficulty of assimilation of digestive products from the gastro-intestinal tract, and further that after the assimilation there is also difficulty with the utilization of the materials, due to thyroidal inactivity. In two cases of this series nitrogen balance was determined, the patients showing definite and prompt recovery after proper treatment, to the extent that food was better assimilated and utilized, as evidenced by the beginning of growth and the laboratory data.

The article further deals with functions of the thyroid gland, and in summary form the authors detail certain conclusions. The picture of hypothyroidism is one of arrested development, degenerative processes of the tissues, lessened food absorption from the alimentary tract, poor utilization of the absorbed digestive products, abnormal carbohydrate metabolism, decreased excretion of purin bodies and the appearance of urinary creatin and general lessening of all metabolic activity—all caused by a hormone hunger due to insufficient thyroid secretion.

PATTEN, Philadelphia.

STEINACH'S OPERATION. G. HOTZ, Schweiz. Arch. f. Neurol. u. Psychiat. **7**:344-349, 1920.

In 1910, Steinach advanced experimental evidence to show that rats in which the male gonads had been removed and replaced by female gonads took

on feminine attributes, and that the reverse occurred when ovaries were replaced by testicles. This applied to both physical and psychic characteristics, which are probably controlled by the cells of Leydig in the testicle and the luetin cells in the ovary. Bouin and Ancel, in 1903, showed that ligation of the vas deferens interrupted spermatogenesis, but led to hypertrophy and hyperplasia of the interstitial cells. Tandler and Gross, in 1908, produced the same effect by roentgen-ray radiation. These animals retained their secondary sexual characteristics and libido.

With this as a basis, Steinach attempted to bring about a regeneration of the involuting gonads by ligaturing the vasa deferentia, later by passing a ligature between the testicle and the epididymis. The aging rats were "rejuvenated" in every respect and sexual activity restored to an unexpected degree. The life of these animals was reported to have been lengthened by seven months, which is about one-fourth. The second senium advanced uninterruptedly, however.

The operation was carried out on man by Lichtenstern by transplanting the testicles of cyptorchids. Steinach's designation of "rejuvenation," *Verjuengung,* precipitated a great deal of comment on the part of the general press.

Hotz was very much interested in this work and reports a case of a 50-year old merchant, who presented himself with the request that this operation be performed on him so that he would be able to carry out his business responsibilities more efficiently. He had noted that his memory and grasp of the business situation had diminished during the past four years, and felt that it would soon be necessary for him to give up his work. The patient was of the nonsmoking, totally abstinent, vegetarian, homeopathic and nature-healing type. The operation was carried out as he requested, no mention being made to him of the questionable value of such a procedure. Six weeks later the operation had its full effect; the patient stated that he could work as quickly as he could ten years previously, that his memory was exceptionally good and his sexual powers increased. On the latter point, however, he laid little stress.

The experience of a younger man is recounted, who had had the operation performed because of failing libido, with absolutely no benefit.

Hotz performed the experiment of transplanting ovaries and testicles into the belly-wall of rats with the view of determining the viability of transplants. Without exception, he found acute necrosis of all testicular tissue; the cells of Leydig, however, persisted somewhat longer, but all of them were necrotic after a period of four weeks. The fate of the ovaries was similar. In previous work he had transplanted the thyroid in cretins with the result that in five months the entire tissue had been organized into a fibrous scar.

Experiments on rats cannot be looked on as the full equivalent of a similar condition in man, as the testicles of a rat are much larger in relation to body-weight, the amount of interstitial tissue comparatively greater and sexual activity more marked. As a matter of fact, the operation of tying the vas in man is not new and has been done for about twenty-five years in order to bring about a reduction in prostatic hypertrophy; the results have never suggested a rejuvenation.

The writer also called attention to the fact that in epididymitis, arteriosclerosis, diabetes, carcinoma, alcoholism, cirrhosis of the liver and other conditions in which atrophy of the spermatogenic tissue occurs, there is an increase of the interstitial tissue without any suggeston of increased vitality.

Tandler and Gross have shown that in moles there is a decrease of interstitial tissue coincident with increased spermatogenesis during the mating season, and an increase during the period of diminished spermatogenesis.

The conclusion seemed apparent to Hotz, therefore, that atrophy of the spermatogenic tissue is primary. Lessening of tissue pressure follows, which in turn leads to a compensatory hyperplasia of the interstitial tissue. He also had the opportunity to observe a patient in whom an epididymectomy was performed on account of tuberculosis on the left side five months previous to a similar operation on the right; a careful study of the testicles showed absolutely no difference on the two sides.

The writer feels, therefore, that he cannot indorse the views of Steinach and Lichtenstern. He is convinced that Steinach's operation is valuable only as a powerful suggestion.

WOLTMAN, Rochester, Minn.

A CASE OF ENCEPHALITIS LETHARGICA INVOLVING CHIEFLY THE CEREBRAL CORTEX. G. A. WATSON, Neurol. & Psychopath. **1**:1 (May) 1920.

The case reported is that of an anemic woman, aged 28. Clinically she presented a picture of agitated melancholia, having attempted suicide by cutting her throat a few days before admission, and after admission by trying to tear the wound open. She remained in this mental condition without change for four months. Then for three days she seemed to be very ill and vomited several times. During the ensuing two months she seemed to improve mentally, but, at the end of this time, began to complain of pain in the back and down the left thigh, and she limped when walking. She was confined to bed. This condition continued for two months, then she lost her power of speech and the right side of the tongue became paralyzed (eight months after admission). She was drowsy, disinterested and "lethargic," but could be aroused to answer questions. Eleven days later there was a right hemiplegia involving the face, arm and leg, with anesthesia and analgesia including the right half of the tongue. The tendon reflexes were especially brisk on the right, although the right plantar and abdominal reflexes were absent. Optic neuritis of moderate degree was present. A week later marked atrophy of the right arm, forearm and hand and of the left calf and thigh were noted. She died the following day. During the illness her temperature was subnormal, and the blood count showed a definite leukocytosis.

At necropsy the brain was found to be soft, and in the left hemisphere there was a large area of recent softening with adherent membranes. After hardening, the sectioning of the area disclosed streaks of vascular congestion that was not grossly apparent in the rest of the brain, but no hemorrhage. Microscopically the membranes showed increase of vessels with marked congestion and here and there small hemorrhages into and beneath the membranes. In the area of softening there was an enormous increase of vessels with marked cellular proliferation, mainly around the vessels, but in the most affected parts spreading widely into the surrounding tissue. The proliferative elements were lymphocytoid, plasma and pseudoplasma cells, but mainly polyblasts, epithelioid and reticulate types of cells, obviously originating from the adventitia. Neuroglia cells were somewhat increased, and the endothelium of the vessels showed proliferation. Small hemorrhages were found around the vessels, though they were not numerous except in the putamen and precentral convolution, where they were rather large. Many veins showed partial occlu-

sion by thrombi. Around many vessels were rarefied sievelike spaces—appearing like an exudate—especially in the white matter, and containing amorphous deposits and proliferated cells which stained well. Nerve cells were affected in the softened area corresponding to the patchy intensity of softening. The precentral and subfrontal regions, the insula, putamen and part of the temporal lobe were mostly involved. The putamen seemed to have been most affected showing intense vascular and cellular increase with destruction of cells, hemorrhages, perivascular sievelike areas and chromatolysis, the remaining portions of the basal ganglions being little involved. In the temporal region the process appeared to be of longer standing and not as acute. In other parts of the brain there was some increase in vascularity, and the vessels showed some adventitial proliferation, increase of glia cells in the vessel vicinity and general cell destruction—although nowhere was the process as marked as in the softened left hemisphere. The medulla and pons showed thickened membranes which were infiltrated, the vessels congested and containing hyalin thrombi; occasionally there was a small hemorrhage. There were adventitial proliferation and glia cell increase and much infiltration with round cells, most marked in the medulla. The cord showed similar changes of the membrane and vessel and small, round-cell infiltration in the gray and white matter. The nerve roots and ganglions evidenced congestion, cellular proliferation and in the ganglions small hemorrhages.

The condition is termed a meningo-encephalo-myelitis on account of its widespread distribution, but the chief stress occurred in the left hemisphere in the areas mentioned.

PATTEN, Philadelphia.

STATISTICS OF EPIDEMIC ENCEPHALITIS. I. S. WECHSLER, Neurol. Bull. 3:87 (March) 1921.

Wechsler obtained accurate information concerning 864 cases of epidemic encephalitis, from private as well as hospital records. Although the entire country had fair representation, more than half of the cases were gathered from New York City.

The age incidence ranged from 4, 6 and 7 weeks to 84 years, the greatest number of cases occurring between the ages of 20 and 50. The period of childhood and adolescence was much less affected than it was in the recent poliomyelitis epidemic. Males were attacked much more severely than females, the proportion being three to two. Practically every occupation was represented. Seventeen physicians, almost 2 per cent. of the group, were included. Fifty-four per cent. of the patients were foreign born. As most of the cases occurred in adults over 25 years of age, the majority of the patients were married. Pregnancy was a complication in twenty-two cases reported. Four of these went to a fatal termination. Wechsler emphasizes the low mortality in this type of disease compared with that of influenza when complicated by pregnancy.

Regarding familial incidence: In five instances the disease occurred in two members of a family. In view of the practical absence of contagion in hospital cases (although two interns were among those reported) direct transmission is considered a negligible factor.

Wechsler found a fairly low mortality rate. Of 850 patients, 178 died, a percentage of almost 21. Death occurred chiefly in those presenting psychotic, delirious and meningitic symptoms with high fever and acute onset.

The majority recovered more or less completely, but many manifested sequelae, especially those of the parkinsonian type, and those having ocular disturbances, tremors, abnormal involuntary movements, etc.

Under the heading of clinical forms and anatomic localization, over twenty types of the disease are included. The lethargic group leads with a percentage of 35. The parkinsonian type, assuming second place, was present in 15 per cent. of the cases.

McKENDREE, New York.

A CONTRIBUTION TO THE STUDY OF GLIOMA. S. UYEMATSU, J. Nerv. & Ment. Dis. **53**:2 (Feb.) 1921.

This important article opens with a discussion of three basic questions: (1) the differentiation of glioma from sarcoma; (2) the pathogenesis of glioma, and (3) the relation between pathologic proliferation of neuroglia and tumors of neuroglia origin. The author has studied four brains showing gliomatous growths, not only as regards the tumor itself but also the general glial condition in the entire brain.

The first case studied was one diagnosed clinically as paresis. At necropsy a central glioma of the third ventricle was found which on microscopic section proved to be a glioma, and throughout various areas of the brain there was a general increase of glia cells, without perivascular infiltration and no disturbance of the cyto-architecture and myelo-architecture, and no changes in the nerve cells. The changes in the neuroglia consisted not only of increase in the cells but also of a deformation of the normal spheric type of nuclei. These neuroglia changes were most marked in the left cornu ammonis, the right postcentral convolution and both frontal lobes. Histologically no distinction was noted between the tumor and the gliomatous areas. The cornu ammonis did not show increase in volume, making it difficult to classify the process. According to the histology which was identical with the tumor itself, it ought to be called a diffuse glioma or diffuse gliomatosis, but when the gross picture was taken into account it seemed to the author more justifiable to classify it as a diffuse sclerosis.

The second case was also diagnosed clinically as paresis and at necropsy showed a superficial glioma of the left frontal lobe. On microscopic section no evidence of paresis was found. The tumor itself consisted of glia cells and fibers. The cells showed two different types: one small, like the normal glia cells; the other larger, appearing more like ganglion cells of the cortex or the anterior horn of the cord, which showed the characteristics of the granule cells of Merzbacher. A tumor of this nature was designated by Stroebe as glioma gangliocellular. The author suggests the name granule cell glioma or giant cell glioma. This case also showed a marked general gliosis, not only in the cerebrum but also in the cerebellum, pons, peduncles, medulla and cord; not of the same intensity throughout but showing considerable local differences. The pons and the occipital pole of the right hemisphere showed the most marked gliosis. In this case there was found in the white substance of the right occipital lobe a small cavity lined with cubic epithelial cells in places in a single layer, in others 4 or 5 cell layers deep. It was not connected with the lateral ventricle, but in general appearance it was exactly similar and was considered to be a displaced or invaded neural canal.

The third case was that of a superficial glioma of the left temporal lobe; microscopic section showed it to consist of many large cells like those in the preceding case and a smaller number of small spheric cells, like the normal neuroglia cells of the white matter. Through the brain a general increase of

glia was noted, but differing from the first two cases in that the nuclei did not vary so much in shape, being more or less spheric.

The fourth patient whose case was diagnosed clinically as traumatic psychosis had a tumor of the left internal capsule that consisted of more cells than fibers, especially in the center. Where the neuroglia cells were closely packed, epithelial formations of various forms and sizes were encountered, which on higher magnification were seen to show the characteristics of ependymal cells. The neuroglia cells through the brain were increased; the nuclei were more or less altered, normal, spheric cells being rarely observed.

In the summary the author discusses the question of the diffuse gliosis which he found in all of his cases of glioma and absent in other tumor cases, used as control, which were not glioma. The theory of Storch that some stimulus cast forth from the tumor is the proliferative stimulus is not accepted by the author because of the distant gliosis. The second theory of intracranial pressure producing it was ruled out by the absence of gliosis in other tumors. The third theory, that of Ranke, that tumor cells may wander out from the tumor into neighboring tissue is also insufficient. The author's opinion is the assumption of a common causative factor for both general gliosis and tumor formation, such as happens in diffuse sclerosis.

The article is well written, the pictures are good, and an immense amount of work has been done. The general increase in glia throughout the entire brain in glioma, while not new with this author, is brought out forcibly by him.

WINKELMAN, Philadelphia.

ON THE SYMPTOM COMPLEXES OF LETHARGIC ENCEPHALITIS WITH SPECIAL REFERENCE TO INVOLUNTARY MUSCULAR CONTRACTIONS. F. M. R. WALSHE, Brain **43**:197-219 (Nov.) 1920.

The author discusses the remarkable clinical diversity of lethargic encephalitis and suggests that the numerous schemes of classification and description adopted, although inevitable in the early and growing stages of knowledge of so polymorphic a disease, tend to confuse rather than to lend precision to our conception of this disease, for practically every case presents simultaneously, or at some phase of its course, the features of several clinical types. As an example of the ineptitude of purely symptomatic classification, he briefly discusses myoclonic encephalitis.

He suggests that the use of focal nervous signs for the purpose of defining types would insure a uniform terminology in the description of the disease and so obviate much of the confusion in the literature. On this basis, the author contrasts acute poliomyelitis and lethargic encephalitis. He further illustrates the points discussed by seven personally observed cases showing various positive motor symptoms. He draws the following conclusions: 1. "It seems probable that the psycho-motor excitement of the 'choreiform' manifestations of lethargic encephalitis, the jacksonian fits, and the rhythmic fibrillary, the fascicular and muscular contractions collectively grouped by various writers as 'myoclonic' symptoms are in fact the expressions of an irritative or exciting action of the virus of the disease on neurons of the three physiological levels (Hughlings Jackson) of the nervous system, and that such symptoms can be localized on the same principles as are employed to localize negative or paralytic symptoms.

2. "If we accept this view of the irritative origin of the motor symptoms under discussion, a striking feature emerges; namely, that where the virus

produces negative or paralytic symptoms it has, ever since the first appearance of the disease, shown a definite and characteristic selective action on the cells of the basal ganglia and on those of certain motor nerves in the brain stem, producing the familiar basal ganglia and midbrain types of the disease with its associated lethargy.

3. "In its irritative or exciting effect the virus appears to act equally on any and every part of the nervous system, from cerebral hemispheres to spinal roots, hence the polymorphic character of cases showing what we may call irritative symptoms. It is such cases that present the many difficulties in any attempt to classify lethargic encephalitis into clinical types. Possibly we may be dealing with a complex virus in which more than one active component exists."

BLITZSTEN, Chicago.

PSYCHOSIS ASSOCIATED WITH TETANY. A. M. BARRETT, Am. J. Insan. **76**:373 (April) 1920.

A psychosis associated with tetany has rarely been observed. Barrett reports two such cases and gives a good summary of the literature on the subject. In the first case a mental condition preceded the tetany, while the reverse order obtained in the second.

In Case 1 the psychosis followed a domestic sorrow and was characterized by depression, loss of weight and sleep, indicanuria (as high as 100 mg. in twenty-four hours), achlorhydria, periods of stupor, retardation and irritability. Soon after, neurologic symptoms appeared, manifested by spasticity, tremor, increased reflexes, flexion of the elbows and adduction of the thumbs —these positions being more or less constantly maintained. The movements were ataxic. The patient was constipated. The mental unclearness progressed with apprehension, disorientation, irrelevancy, confusion and hallucinosis. There were jerking and twitching movements of the upper half of the body—often brought out quickly by merely tapping any of the muscles. Chvostek and Trousseau phenomena were present. Ten weeks after admission the stupor deepened, and the twitchings became marked. Several days later he complained of pain in the back of his head and arms, and the characteristic accoucheur position was assumed. He then became stuporous and restless by turns, and the tetanoid contractures came on at intervals evidently accompanied by considerable pain. Nearly five months after admission he died in a condition of considerable weakness, but with persistence of the muscular irritability. The necropsy diagnosis was "ascending thrombo-phlebitis of the right iliac and femoral veins; embolism and thrombosis of the pulmonary vessels; purulent pneumonia; emphysema; atrophy and passive congestion of all organs." In the nervous system a large cyst in the anterior lobe of the pituitary gland near the base of the stalk was discovered. The parathyroid glands showed no abnormality on histologic examination. The nerve cells in the cortex showed marked chromatolytic changes and fatty pigmentation.

The second case began with a sudden attack of sharp pains beneath the right patella. Soon the leg dragged and became stiff. The stiffness extended into the thigh and up the back. Bending on arising from a sitting to an erect posture was difficult. The condition grew progressively worse until, two years before his admission, his eyesight began to fail and he fell frequently, lying with all parts of the body in marked flexion. A year later he became mentally unclear, had somatic delusions, was depressed and untidy, confused at times, disoriented, irrelevant and often quite incoherent. Neurologically

an increase and disproportion in reflexes occurred, tremors developed, the tongue deviated to the right and the ocular fundi showed venous congestion. After three weeks he remained mentally clear and was discharged. The muscular irritability continued, however, and was characterized by tonic contractures, tremors and jerking movements. He was readmitted three months after discharge. Except for memory defect, his mind was clear. Neurologic examination at this time revealed increased reflexes in the upper extremities, the Babinski reflex bilaterally, speech defect, sluggish pupils, weak and staggering gait, loss of sense of position in the toes and spontaneous jerkings of the arms and legs at intervals. He then became stuporous. Generalized tenseness and contraction of muscles developed with tremors at irregular times. The Chvostek and Trousseau phenomena were present. The position of the hands and arms was typically accoucheur during the attacks. Death occurred nine days after the second admission. Postmortem findings failed to reveal changes in the parathyroid glands, but the nerve cells in the cortex showed widespread and severe alterations. The neuroglia and blood vessels also shared in the degenerative processes. The areas affected included practically all parts of the central nervous system. The pituitary gland showed many small cysts in the posterior lobe and an exceptionally large one near the base of the stalk.

The author suggests the original disease in the pituitary glands as the causative factor in the altered metabolism producing secondarily the disorder of the nervous system and the dysfunction of the parathyroid glands.

PATTEN, Philadelphia.

PIGMENTARY RETINITIS AND HYPOPHYSIAL OBESITY. CHAILLOUS, Ann. d'Oculistique **158**:100.

The common ocular involvement in disease of the hypophysis is bitemporal hemianopsia. Retinitis pigmentosa, with obesity and polydactylism as a symptom triad, was described by Bardet in his theses of 1920.

The author reports the case of a patient, a girl aged 4 years, who consulted him in 1914, because the parents said her vision was affected. Enormous obesity of the patient was striking, but the eyegrounds were negative.

In 1919, this patient was again seen, and the obesity was even more marked especially of the abdomen and legs, but no points of tenderness were present. The ocular symptoms were increased. Vision was so reduced that she had to grope her way about. Examination of her eyes showed a slight external strabismus but no nystagmus. The pupils were normal. The disks were slightly discolored on the temporal side. At the periphery of the fundus were small patches of choreoretinitis, such as those seen in pigmentary degeneration of the retina. A radiograph of the skull showed the sella turcica to be normal, while Wassermann reactions of both the child and the mother were negative.

Thus, in this patient were found obesity, hexadactylism and pigmentary degeneration of the retina. Pituitary therapy was tried with no success.

A discussion of Bardet's paper follows, which tries to show a relationship between pituitary disease and retinitis pigmentosa. Chaillous cannot agree with his reasoning, from the evidence produced, although he thinks a new field has been opened up in the study of such conditions, in which the radiographer, neurologist and ophthalmologist may well cooperate.

SCARLETT, Philadelphia.

THE TREATMENT OF CEREBROSPINAL FEVER. C. WORSTER-DROUGHT, Neurol. Psychopath. **1**:1 (May) 1920.

Early diagnosis and treatment are of paramount value in this disease. It is not harmful to inject serum when on lumbar puncture the fluid is found turbid. In the premeningitic stage, especially if there are suspicious petechial spots and premonitory signs, intravenous administration of serum is a wise procedure, as it has been conclusively shown that infection occurs through the nasopharynx via the blood stream. Serum can be administered intravenously with safety up to 200 c.c. or more. Early examination of the spinal fluid may disclose the presence of the organism before there is much cellular reaction and before the fluid becomes turbid, hence the advantage of examining smears from centrifugalized fluid. The intravenous administration of serum after meningitis has developed is of doubtful value due to the loss of permeability of the membrane of the subarachnoid spaces. At this stage, however, intraspinal administration is of great advantage since it concentrates the antibodies and immune principles where the bulk of the disease process is found. Furthermore, it has been shown that absorption into the blood takes place quite rapidly from the subarachnoid spaces, and it is best to repeat the injections at least every twenty-four hours. The author uses as a total from 120 to 600 c.c. of serum in his cases, employing the gravitation method of administration since it is much less likely to produce respiratory failure than the syringe method. He advocates the continuance of the injections until decided improvement has taken place clinically and the fluid is free from organisms. After the discontinuance of the serum he continues to drain the intrathecal space, believing it assists in carrying away waste products and increases the inflow of healthy fluid. At the first sign of recrudescence he repeats the serum injections without delay. Vaccines are given; 250 million organisms are injected subcutaneously during the first three days of the illness and the number is increased by 500 million at each succeeding injection (every fourth day) up to a maximum dose of 2,500 million. Polyvalent vaccines are best while waiting for the determination of the type of meningococcus and the preparation of autogenous vaccines. The polyvalent vaccine represents strains of all four types based on the Gordon classification.

As far as general management and treatment are concerned, besides the usual things, the author advocates keeping the patient's head low, changing from a liquid to a semisolid diet as soon as possible, administering phenacetin and caffein for headaches, acetyl-salicylic acid for the muscular pains and hypnotics for sleeplessness. Reinfection through the nasopharynx is prevented by swabbing with chloramin-T (1 per cent.) as soon as possible after the onset of the disease.

Of seventy-two patients treated by the author as outlined, fifty-seven recovered and fourteen died. Thirteen of the fatal cases were of the fulminating type, acute fatal, or progressively purulent types. Of the fourteen who died only one had internal hydrocephalus, having been admitted three days after a recrudescence set in. The average duration of illness of those who recovered was fourteen days, and only two or three gave evidence of symptoms of internal hydrocephalus during the disease.

PATTEN, Philadelphia.

THE POSTENCEPHALITIC PARKINSONIAN SYNDROME. J. A. BARRE and L. REYS, Bull. méd. **35**:351 (April 27 and 30) 1921.

The parkinsonian syndrome has been relatively frequent in the course of epidemic encephalitis. In a series of 101 cases which the writers have studied,

it developed in twenty. They emphasized that, contrary to other reports, the parkinsonian syndrome sometimes accompanies or follows a myoclonic encephalitis.

They review first the symptoms which are common to Parkinson's disease (paralysis agitans) and to the parkinsonian syndrome (referred to as the pseudoparkinsonian syndrome) which develops in certain cases of epidemic encephalitis.

In the pseudoparkinsonian syndrome the muscular rigidity has a predilection for, and reaches its maximum in, a cervicofacial distribution. Likewise, tremor in the pseudoparkinsonian syndrome is most often seen in the face.

Slowness of voluntary movements and absence of spontaneous movements is present in both. Although this slowness of movement almost always is accompanied by rigidity and tremor, it has been observed apart from these.

Disturbances of equilibrium, vertigoes and nystagmus occur in both. They are moderately rare in true Parkinson's disease and much more frequent in the postencephalitic syndrome. The acuteness of the developing infectious lesions explains this prominence in the latter. The oculomotor signs, so outstanding in the pseudoparkinsonian syndrome, are not totally unknown in the true. The vasomotor disturbances appear, to the writers, to be the only features peculiar to Parkinson's disease.

The encephalitic parkinsonian syndrome never appears insidiously or without having been preceded by other pathologic manifestations generally of an infectious order. With this point in mind, it is possible to go further and distinguish three types of onset of the syndrome. 1. It constitutes an initial manifestation and dominates the encephalitis. 2. It develops as an immediate sequel of the encephalitis when the other manifestations of the latter still are evolving. 3. It appears during the convalescence.

The concluding portion of the paper deals with therapy. The writers recommend scopolamin and atropin used in alternation. The latter is used in a dose of 1 cg., given two times a day. Atropin administered in this way can arrest the evolution of the parkinsonian phenomena and produce a durable amelioration.

DAVIS, New York.

MUSCULAR INFANTILISM. ALEXANDER GIBSON, Arch. Int. Med. **27**:338 (March) 1921.

Gibson reports a case of unusual interest. In the family history of the patient there was definite hereditary evidence of similar conditions of muscular weakness. Of twenty-six males, fourteen were affected and of twenty-one females, seven were affected. The patient was unable to perform any unusual physical exertion from childhood to the date of his admission to the hospital at the age of 26. He got along very well in his work and could even play golf without discomfort, but anything more strenuous, like rowing or playing baseball, was impossible. On rising from a sitting position to an erect posture, he required the aid of his hands and arms. His general health and mental ability were always good. Physical examination revealed: height, 5 feet 8 inches; weight, 155 pounds; musculature small and poorly developed; distribution of fat rather in excess over the upper part of the trunk, over the abdomen and about the hips and thighs. His contour suggested the female type, with tapering extremities and small hands. The blood pressure was from 130 to 140 systolic; the blood picture was practically normal; roentgen-ray examination was negative. His bones were small. The Wassermann reaction

was negative. There was no sexual impotence (nor was there any history of disturbance of reproductive power in the family).

He was given various organic extracts, some drugs and certain muscular exercises were prescribed. Raw suprarenal extract caused gastric instability and had to be discontinued. Graduated exercises caused no improvement. Thyroid extract made his general condition worse if it affected him at all. Pituitary gland feeding caused no improvement. Strychnin met with no response. His strength is well illustrated by a comparison made with a laboratory attendant who could do 400 kilogram cm. of work in two seconds with the second finger of the right hand, while the patient could accomplish only from 12 to 15—a percentage of the normal of 4 or 5. Microscopic examination showed a piece of muscle to be essentially normal.

Careful examinations of the urine were made in the laboratory, as well as chemical tests on blood. The conclusions arrived at were that the creatinin excreted suggested the actual extent of muscular power which he possessed. Creatin excretion suggested an infantile condition, and varied little on a meat diet. Blood sugar and sugar utilization were normal. On the basis of creatin in the urine (which is normally absent after maturity) and the small muscular power possessed, the author places the condition in the nomenclature of muscular infantilism.

PATTEN, Philadelphia.

THE FATE OF THE NEURAL CREST IN THE HEAD OF THE URODELES. F. L. LANDACRE, J. Comp. Neurol. **33**:1-43 (April 15) 1921.

Since Miss Platt's demonstration, in 1894, of the contribution of ectoderm to the formation of cartilages and other mesodermal structures in amphibian embryos, the origin and significance of this mesectoderm (i. e., mesoderm of ectodermal origin) have been subjects of much study and some controversy.

Landacre, in salamander embryos, has been able to follow the development of the ectodermal cells which form the neural crest of the head up to late stages when the tissues have acquired their definitive characteristics. In urodeles the neural crest is first incorporated into the neural tube, then is detached and rapidly extended ventrally. From the dorsal portion the general cutaneous and general visceral components of the fifth, seventh, ninth and tenth cranial ganglions are formed, together with some loose mesenchyme. The ventral portion of the continuous neural crest sheet of cells extends downward ultimately to the ventral surface of the head and gives rise to loose mesenchyme and to a number of the cartilages of the head, namely, the anterior portion of the trabeculae, Meckel's cartilage, the palatoquadrate bar and all of the branchial cartilages except the second basibranchial. The greater part of the mesenchyme in the ventral head region and in the branchial region is of ectodermal origin. The remaining mesodermal structures of the head (muscles, connective tissues, cartilages, etc.) are of entodermal origin.

The dogma of the specificity of the germ layers is thus apparently broken down. The significance of this two-fold origin of the mesoderm is not clear. Speculations regarding its possible relations to the origin of sympathetic ganglion cells, chromaffin tissue, neurilemma sheath cells, etc., must await on further observations.

C. J. HERRICK, Chicago.

SINISTRALITY IN RELATION TO HIGH BLOOD PRESSURE AND DEFECTS OF SPEECH. CLARENCE QUINAN, Arch. Int. Med. **27**:255 (Feb.) 1921.

The subject of sinistrality is taken up from the standpoint of heredity, prevalence and symptomatology. The author agrees with Rameley whom he quotes as follows: "The condition is a Mendelian recessive and probably exists in about one-sixth of the population." From recent statistics, it seems that about 4 per cent. of normal people are left-handed. In regard to the "master-eye" and sinistrality, one writer is quoted as finding 57 per cent. right-eyed persons who were left-handed. Speech defects are common in sinistrals as compared with dextrals, probably because there is more difficulty in finely coordinated movements in the former class. Also, sinistrality is exceptionally prevalent among the mentally defective. The author suggests as explanation that there is a relative inferiority in the organization of the central nervous system.

With regard to the blood pressure in sinistrals, a survey of 600 men was made living under the same conditions, the majority being over 60 years of age. Of this number, forty-two were sinistrals, and twenty-eight of these were left-handed, while fourteen were right-handed sinistrals (that is, those who by training have become right-handed but are by nature left-handed). Fifty per cent. of the forty-two were right-eyed and 50 per cent. left-eyed (classified according to the "fixing eye"). The fourteen right-handed sinistrals were left-eyed. For further classification the forty-two were divided into three groups: (1) left-handed, left-eyed (pure sinistrals); (2) left-handed, right-eyed (crossed sinistrals); (3) right-handed, left-eyed (crossed sinistrals). For comparison with sinistrals, blood pressure records were made on a group of 100 unselected right-handed, right-eyed dextrals, and for tabulation purposes this group is called Group 4 by the author. Five men in Group 4 as well as five men in Group 1, and two men in Group 2, gave a history of stammering. The average blood pressure values in the four groups were: (1) 166.7, (2) 182.1, (3) 174.9, and (4) 154.5 mm. This shows a percentage of from 71 to 85 of high pressures in the first three groups as against 46 per cent. of high pressure in Group 4. The percentage of stammerers in the four groups was: (1) 35.7, (2) 14.2, (3) 0, and (4) 5.

The author concludes that "high arterial tension occurs more frequently in left-handed than in right-handed people, and that it is suggestive of constitutional inferiority. Stammering occurs in sinistrals with a frequency of from three to seven times greater."

PATTEN, Philadelphia.

A MYOCLONIC TYPE OF MYELO-ENCEPHALITIS OF MALARIAL ETIOLOGY. M. G. MARINESCO, Ann. de méd. **9**:1 (Jan.) 1921.

The writer first gives a clinical description of a case, in which fever, widespread myoclonus, prostration and death on the twenty-fifth day were the outstanding features. There were 10 cells in the spinal fluid. The case was considered as epidemic encephalitis until a subsequent blood smear showed the presence of malarial parasites.

The necropsy report, which is given in detail, establishes the etiologic significance of the malaria. In brief, the chief pathologic findings were the presence in the central nervous system of small so-called malarial nodules. Measuring from 210 to 132 microns, their histologic constitution depends on a leukocytic element combined with a cellular element of vascular origin. Cells

carrying pigment are present and in some instances cells bearing the parasite. The presence of the latter and of the pigment justifies the term malarial nodule.

Marinesco considers the essential cause of their genesis to be a formative irritation of vascular origin.

Their distribution included the cerebral cortex, the peduncles and bulb and both the gray and white substances of the cervical and lumbar cords. The cerebellum had been invaded by them only to a slight degree and the spinal ganglions to still a less extent.

The writer points out especially that the nodules were not associated with hemorrhages. The latter were encountered in areas free from nodules.

Though the involvement of the central nervous system was widespread, necropsy study failed to demonstrate malarial parasites in either the spleen, the liver, the suprarenals or ovaries.

DAVIS, New York.

ADMINISTRATION OF A PITUITARY EXTRACT AND HISTAMIN IN A CASE OF DIABETES INSIPIDUS. R. B. GIBSON and F. T. MARTIN, Arch. Int. Med. **27**:351 (March) 1921.

The case is that of a man, 24 years of age, who complained of loss of appetite, pain in the back and abdomen, occasional dimness of vision, coldness and chills, polydipsia, polyuria, general weakness and loss of weight. The onset of these symptoms was indefinite and the polyuria could not be traced farther back than 1914, when he contracted syphilis. The bearing that syphilis has on the condition could not be definitely determined. The physical examination revealed no evidence of pituitary disease, except that the sella turcica was enlarged; this was not interpreted as abnormal. The blood pressure was: systolic, 104; diastolic, 62. The spinal fluid Wassermann reaction was negative, but the blood showed a ++++ reaction.

The laboratory work carried out in the case consisted of thorough studies of metabolism, with frequent examinations of the urine. At the time of admission the volume of urine varied from 15 to 20 liters per twenty-four hours and had a persistent low specific gravity. Hypodermic injection of pituitary extract (obstetric pituitrin) gave a response in a short time, but in reality the effects were fleeting. The ingestion of the tablet preparations gave a similar response, but its effects were more enduring. The total volume of urine was lowered to 10 liters or less per twenty-four hours, and the nitrogen output was considerably decreased. The percentage of uric acid was high, however (conforming to the reports of Hammett, Patten and Suitsu on nonendocrine cases). The nitrogen partition in general was normal; glycogenesis was not reduced, but there was a hypoglycemia.

Histamin injections produced results somewhat similar to the pituitary injections, but were much less effective. The systemic reactions to the preparation were so severe (intense headache and general prostration) that it was discontinued promptly. The authors conclude that histamin is not the active principle of the pituitary gland.

The general conclusions were that pituitary substance exerted only a temporary effect on the polyuria, but during its activity appreciably diminished the volume of urine, decreased the nitrogen output and increased the uric acid percentage.

PATTEN, Philadelphia.

Society Transactions

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Monthly Meeting, April 21, 1921

JAMES B. AYER, M.D., *in the Chair*

IS THE TREATMENT OF PATIENTS WITH GENERAL PARALYSIS WORTH WHILE? DR. HARRY C. SOLOMON.

Opinions differ as to whether or no it is worth while to treat patients with general paralysis. To distinguish even pathologically between cerebrospinal syphilis and general paralysis is difficult. Cases that apparently are cerebrospinal syphilis prove to be general paralysis or vice versa. For a diagnosis of general paralysis, in addition to typical clinical findings there should be certain laboratory findings: a Wassermann reaction which is positive with 0.2 c.c. of spinal fluid, a paretic gold curve, globulin, excess albumin and lymphocytosis about 100. Practically all of the nine patients shown met these requirements; their treatment had consisted largely of the intensive use of arsphenamin, 0.6 gm. twice a week over a period of three or four months.

In the first case treatment was begun in 1916. The patient left the hospital after two months of treatment, apparently having clinically recovered. Her spinal fluid was practically negative when examined in 1919. It is now positive, but she has been clinically well for a period of five years.

The second patient was a man of 53, who had been admitted to the hospital with a diagnosis of general paresis in 1915. During the first four months he received a great many treatments with spinal fluid drainage. When last examined, January, 1921, he had a negative Wassermann reaction, a cell count of 7 and the colloidal gold curve was 0001100000. He is now making twice the salary he made when admitted.

The third patient did not do well under intravenous injections. He was admitted in April, 1916, aged 37. After two months' treatment with arsphenamin and mercury he was no better, and he was then given three intraventricular injections followed by seven intravenous injections of arsphenamin. On November 16 the spinal fluid was negative. The fourth patient was sent to the hospital because he had become inefficient. On March 14, 1919, he had a shock and felt dizzy when he got out of bed. His memory had been poor since then and he had speech defect and writing disorder. He has received both intravenous and intraspinal injections, and although his spinal fluid is not quite negative, he is back at work and is as efficient as he was in his best days.

The six other patients showed similar results. Dr. Solomon thought that if these patients had not been treated, they would have died or would still be at institutions instead of living for a period of from two to six years in good health and with good efficiency.

DISCUSSION

DR. ARTHUR H. RUGGLES said that cistern puncture had been used about fifty times at Butler Hospital with no bad results and with the most satis-

factory cooperation on the part of the patients. They seem to have less reaction following this treatment than after the intraspinal method, and it presents a new point of attack in the case of patients who do not respond either to intravenous treatment plus spinal drainage or the combined intravenous and intraspinal treatment.

Dr. Solomon said he could not give the exact figures for the number of remissions in the patients treated. Some were treated only a short time because it was impossible to get cooperation. In 1916, of all the patients treated in the state hospitals 25 per cent. were allowed to go out on remissions. Age does not seem to play an important part. Patients treated at 65 years of age have done very well. It is impossible to say why certain persons do well and others badly. A young man with a good heart and kidneys may do poorly, possibly because of an overwhelming infection that has used up his immunity.

Regarding Dr. Ayer's method of cistern puncture, he thought it a practical addition to the treatment of these cases. It is revolutionary in the treatment of brain and cord inflammatory conditions and will greatly alter prognosis.

Dr. Solomon also advocated intravenous injections, if intensively continued, and considered one of his chief mistakes to have been overconservatism in giving no more than 0.6, 0.8 and 1 gm. to a patient. The worst that could happen as a result of too large a dose would be to shorten the life of a patient with only a few months to live, and it has never been shown that larger doses damage or approach the tolerance of the patient.

ENCEPHALITIS WITH SYMPTOMS INDICATING DISTURBANCE IN THE REGION OF THE PITUITARY GLAND. Dr. Douglas A. Thom.

Dr. Thom presented a young girl, born in 1902, who had never been sick until February, 1920. The family history was negative. She graduated from high school at the head of her class two months before the onset of her illness and took a position as a stenographer. Up to the time of her illness she was normal in every way, was not hypersensitive and had no character twists. On Feb. 4, 1920, she dreamed of an automobile accident in which her brother was hurt and she was killed. All the following day her mind was filled with the content of this dream. She went home anxious and worried to find that her brother was all right. From that time on she was restless and was unable to sleep for about a week. Then she slept continuously for four or five weeks. At the beginning of this drowsiness she had diplopia and strabismus. The drowsiness has persisted. She has been emotionally unstable since that time. At the present time she is well-developed, highly cooperative, talks rapidly without much expression, appears to be very intelligent and has perfect insight into her condition. She is strong and well but has a masklike expression; she is otherwise normal. The physical examination is negative except that the hands are held in the parkinsonian attitude, the shoulders droop slightly forward, and she is conscious of a tendency to fall forward. The Wassermann reaction is negative. She suffers from extreme thirst. While at home she drank ten quarts of water daily and excreted about the same amount of urine. This condition has persisted for about a year, dating from a few weeks after the onset of the lethargic condition.

The problem is whether the pituitary or neighboring region has been involved which would account for these symptoms of diabetes insipidus.

ENCEPHALITIS WITH PROMINENCE OF HALLUCINATORY SYMPTOMS. Dr. L. J. Thompson.

Dr. Thompson reported two cases of encephalitis with hallucinations unaccompanied by delirium. Hallucinations in encephalitis have been noted before, but in almost every case have been credited to the delirium. In these two cases, however, there has been no delirium. Of the eighteen patients with definite encephalitis with hallucinations treated at the Psychopathic Hospital, all had delirium except two, and one other.

The first patient was a woman, 37 years old, married, whose symptoms began in November, 1919, with dizzy spells; once she fell. Shortly after she began to have severe, transient, occipital headaches. In January, 1920, she began to have involuntary movements of the legs, arms and head, which shook continuously, so that she could hold nothing, and could neither dress nor feed herself. One month before entering the hospital, a year ago, her psychotic symptoms began. She became suspicious, and said that the people upstairs were taking moving pictures of her which were to be in the newspapers; that they were boring holes in the ceilings and pouring skunk oil through them; and that men were coming through the window. Several times she thought she saw her deceased husband; she said that there were soldiers around the bed. At one time a sword came up beside her bed and disappeared. At the hospital she continued to show hallucinations in all spheres.

Examination showed ptosis of both eyelids, irregular pupils and dancing movements of the eyeballs. There was no definite disturbance of reflexes, but general muscular hypertonus. The spinal fluid showed increase of globulin, 27 cells, a gold curve of 2334321000 and a negative Wassermann reaction. The hallucinations continued for a week or ten days and then suddenly disappeared; she had good insight. At that time the Parkinson syndrome began to increase. She had slight tremor of the hands, a masklike face and dysphagia increased. She left the hospital but still shows residual symptoms.

In the next case there may be some doubt as to the diagnosis. Last July slight shaking of the head was noticed. About three weeks later she said that certain people began to talk about her. Later she had definite hallucinations and was sent to the hospital. She had had headaches for a long time. The spinal fluid showed increase in globulin, 7 cells, and a negative Wassermann reaction. A recent lumbar puncture was negative. After being in the hospital a little while she developed ptosis of the left lid. The knee jerks were unequal. The patient has continued to have definite auditory hallucinations, to which she occasionally reacts by outbreaks of temper.

Dr. Percival Bailey said that during the past year, with Dr. Fritz Bremer, he had made a series of experiments on dogs which showed that the polyuria which often accompanies operations on, and diseases of, the pituitary gland is due to a lesion of the hypothalamus. The patient presented by Dr. Thom had a definite postencephalitic syndrome, but with polyuria excluded there were no pituitary symptoms. That polyuria is due to a brain lesion has been insisted on by Ochsner, Camus and Roussy, Houssay, and Leschke. Recently Harve has described the pituitary glands from several cases of epidemic encephalitis, and one familiar with the structure of that organ would recognize in his description nothing which could not be found in any pituitary gland. Recently, also, Maranon has described cases in which polyuria, adiposity and genital dystrophy have followed epidemic encephalitis.

Dr. Benjamin T. Burley said that in one instance only had he seen several people living in the same house attacked. He mentioned a case closely resembling the one reported by Dr. Thompson. The husband of this patient had died six months before in Worcester. He had been tapped, and the fluid was bloody. The patient had a fluid of the same content. A 16-year old son was taken sick four days after the mother, and he lived only seven days.

Dr. L. J. Thompson mentioned a necropsy case of encephalitis in which the patient had had visual hallucinations. Little was shown pathologically. There have been no cases at the hospital which might indicate contagiousness.

PRESENTATION OF A CASE OF PSYCHOSIS. Dr. C. Macfie Campbell.

Dr. Campbell demonstrated a patient with a psychosis, simple from the point of view of a mechanism, poorly represented in the textbooks and difficult to classify.

The patient was a girl of 24, who was well on March 3. On that day, at her work in a factory, another girl told her a great many things "a single girl should not know." She began to worry and felt queer, emotional and dizzy. She went out, was in a very excited state, wanted to take off her clothes and felt that she had to demonstrate her purity. She was brought to the hospital where she showed some knowledge of her whereabouts. All that night she stood up and looked out of the window; said God was cremating her and that she did not want to die in black. During the next four days she was very much disturbed; by the end of the week the excitement had passed away, her mind was clear and her convalescence had been uneventful.

The mechanism of the case seemed simple. The girl lived a quiet, reserved life, was very critical of approaches by any young man, lived rather more strictly than those around her, and was evidently very sensitive to the sexual topic. The conversation of another factory worker inflamed her imagination, and in the psychosis she lived out a drama of being kidnaped as a white slave. She saw the world in a distorted way and had odd fragmentary delusions, some of which were difficult to interpret. A year ago this girl had been rather upset by an Italian who was making advances to her. She came home very much excited and was emotional for two or three days. Then she passed into a condition in which she spoke little, and for two weeks she was looked on as a sick girl. She did not go to work for six weeks.

The case was a recurrent psychosis, not a simple hysterical delirium of the wish fulfilment type; there were a few elements of rather odd symbolism. Schizophrenic features were present, but the condition was benign and did not pass into a condition of deterioration.

The advisability of trying to make the patient realize the significance of the attack was discussed. This is a type of disorder which plays a large part in practical psychiatry, but which is, as a rule, discussed in a very inadequate way.

DISCUSSION

Dr. A. W. Stearns said that lately he had been much interested to note the ingenuity with which earlier psychiatrists used the assigned cause in diagnosing mental diseases. It is a question whether modern writers are going to prove as ingenious as their predecessors in making use of such material.

Dr. C. M. Campbell stated that he had not attempted to bring out the whole etiology. All that was known was that the girl was earning her living and working well until she left that morning for the factory. Naturally, the efficient cause of such a psychosis is a complicated group of forces. The patient's general efficiency was about that of her social group.

Tentative Classification of Behavior Disorders Used at the Psychopathic Laboratory, Massachusetts Reformatory, Concord, Mass.

	Responsibility	Manifestations	Groups	Diagnoses
Behavior disorders	Annulled	Alieniations	Psychoses	.....
			Epilepsies	.....
		Deficiencies	Intelligence defects	Idiot Imbecile Moron Subnormal
	Limited	Aberrations	Psychoneuroses	.....
			Constitutional psychopathies	.....
			Neurological disorders	.....
			Endocrinopathies	.....
	Entire	Habits of action	Characterial deviations or rectitude	

Diagnoses	Super-normal	Normal or Adult	Sub-normal	Institutional
Psychoses				
Recoverable	.....	.....	.....	.....
Recurrent	.....	.....	.....	.....
Chronic without deterioration	.....	.....	.....	.....
Chronic with deterioration	.....	.....	.....	.....
Traumatic	.....	.....	.....	.....
Alcoholic	.....	.....	.....	.....
Syphilitic	.....	.....	.....	.....
Senile	.....	.....	.....	.....
Arteriosclerotic	.....	.....	.....	.....
Brain tumors	.....	.....	.....	.....
Myxedematous	.....	.....	.....	.....
Epilepsies				
Grand mal	.....	.....	.....	.....
Petit mal	.....	.....	.....	.....
Jacksonian	.....	.....	.....	.....
Masked, etc.	.....	.....	.....	.....
Intelligence defects				
Idiot	.....	.....	.....	.....
Imbecile	.....	.....	.....	.....
Moron	.....	.....	.....	.....
Subnormal	.....	.....	.....	.....
Psychoneuroses				
Hysteria	.....	.....	.....	.....
Psychasthenia	.....	.....	.....	.....
Neurasthenia	.....	.....	.....	.....
Paranoid personality	.....	.....	.....	.....
Constitutional psychopathies				
Coordination psychopath	.....	.....	.....	.....
Sex psychopath	.....	.....	.....	.....
Neurological disorders				
Neurosyphilitic	.....	.....	.....	.....
Tremors, tics, chorea	.....	.....	.....	.....
Endocrinopathies				
Characterial deviations *				

* Specified in case notes and there associated, contrasted and illustrated. Certain observed deviations of frequent occurrence are the too egocentric, lacks self-respect, sex conflict, antisocial, lacks definite ambition, acquisitiveness unchecked, too liabile emotionally, unamenable to reason or authority, etc.

A METHOD OF PERSONALITY DIAGNOSIS AND EVALUATION WITH PROVISION FOR SOCIAL SERVICE PROPAGANDA. Dr. Guy C. Fernald.

Recent advances in the study of defective delinquents owe much to the findings in the field of character, that component of mentality which connotes the quality thereof, in contrast to its degree, i. e., intelligence. Those mental

organizations eventuating in action or behavior are quite as significant as indexes of personality efficiency as are those mental organizations which eventuate in thought and its expression. In actual daily usage and in judicial procedure each personality is held accountable for his behavior, the product of character chiefly, and not his thinking, the product of intelligence.

Characterial deviations and rectitude may not be, as yet, technically tested and numerically scored but are susceptible of presentation by scientific description as are the findings of psychiatrists to the insane. The three fields of inquiry are mental disease, intelligence and character.

The outline is susceptible of such modifications as are required for adaptation to a given group and is submitted for its possibilities rather than as a compendium.

No form of presentation is complete which does not recognize the limitations of categorization. Search must be made in the character of a person for the strength of will needed for overcoming tendencies to sloth, which often defeat the success of those of high as well as those of a lower degree of intelligence. Failure, sociologically and economically is at least as often due to mediocrity of ability to continue to pursue a well selected purpose as to paucity of academic knowledge of the course to pursue. Of two imbeciles incapable of self-determination the one living happily and usefully on a farm as chore boy, trusted within limits but always supervised, the other of no higher grade of intelligence, a tramp who will beg or pilfer but will not work; the one does as well as he may, while the other does not. The essential difference lies in the field of character.

The psychiatric personality investigator's method of choice, then, is first to determine the presence or absence of mental disease, then to canvass the mentality for intelligence age level and for the determination of character deviations and rectitude, the last to be presented in the form of scientific description.

DISCUSSION

Dr. William Healy said that he was glad that Dr. Fernald was emphasizing the study of character as a thing by itself. Even in the study of persons from the standpoint of vocational possibilities, mental tests, which are nowadays so much advocated, are by no means sufficient. As a well-known silk manufacturer said, mental tests, except for clerical workers, did little in the way of giving him useful information in his large factories. And recently a paper has been published, based on investigations in this factory, in which it comes out clearly that there is little correlation between the behavior prognosis, that is, the success at work, and the results on mental tests in the factory at large. Some of their most reliable workmen are people of poor mental capacity. Indeed, it would seem that one would have to be pretty dull to be thoroughly happy pasting labels on boxes for eighteen years. Along this line was a case that Dr. Healy saw years ago. A young man appeared in court accompanied by his employer, who was anxious to pay the fine for a slight delinquency. He explained that the offender was the only one he had ever had who did his particular bit of work satisfactorily. The young fellow turned out to be definitely defective. The employment was in a laboratory, keeping things clean. Thus, there are necessary phases of industrial life in which intelligence is even a handicap to good performance. Character and temperament have a great deal to do with both good conduct and bad conduct. The point of view of Aristotle cannot be assented to; namely, that good intelligence would prevent a man from doing wrong.

But another trouble is that in making studies by means of so-called intelligence tests, all the elements of intelligence are not considered. This is coming out clearly in a symposium on intelligence tests that is appearing in the *Journal of Educational Psychology*. It seems to have taken psychologists a considerable time to grasp this point.

Dr. Fernald's categories are decidedly useful for a practical view of the situation, but Dr. Healy feels that the idea of responsibility is altogether too difficult to be placed among the categories. It is not open to exact thinking. What is responsibility? How can it be measured and recognized? It is a metaphysical conception which cannot be clearly defined, and it has nothing to do with the practical handling of many cases. It is time that psychologists and psychiatrists taught the practical issues of this question plainly to the legal profession. The law attempts to demand the answering of questions that cannot be answered.

Dr. Fernald, in closing, remarked on the importance of character in the life activities of an individual. Defective intelligence is stationary. It must be taken as it is and cannot be improved on, but character growth can be stimulated and improved as may be observed in social service endeavor.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Monthly Meeting, May 19, 1921

James B. Ayer, M.D., *in the Chair*

An unmarried man, 30 years of age, whose family history was negative, had been well up to the time of the present trouble, with the exception of having gonorrhea in 1917. A Wassermann test made some time subsequently was negative. After his enlistment in 1917 he took long hikes in intensely hot weather. They were very exhausting and caused pain and numbness in the anterior portion of his right leg and in the right little toe. The pain and numbness would disappear on resting, but the leg slowly became weaker. After ten months, he had some sort of infection after which the leg was very weak. There had appeared a superficial scar about the size of a silver dollar, reddish blue in color. He was discharged in January, 1919. By this time the scar had spread somewhat and was numb, but not painful. He went to work on a buffing machine which created intense heat, and he noticed that when it touched the scar no heat was felt. The sensation of cold was probably also lost. At present the scar had spread considerably, the anterior thigh and leg muscles were weak and the right anterior thigh numb. There was pain in the anterior tibial group. There had been a suggestion of the same phenomena in the left leg.

The patient was well nourished and unusually muscular. The gait was not remarkable. The Romberg sign was absent. The pupils, fundi and facial muscles were normal. Lateral movements of the eyes caused two or three nystagmoid oscillations. The tongue, soft palate, spine, and muscles of the upper extremities, back and torso were normal. The patient said that the strength of the right upper extremity had diminished. The heart was normal. There was no atrophy of the lower extremities. Occasional fibrillation was observed in a very small area in the anterior tibial region. The general strength of the extremity was not commensurate with the muscle volume. All

forms of sensibility were preserved except over the lower anterior aspect of the right thigh. Here, in a region roughly 3½ by 4 inches, was a keloid formation somewhat red and traversed in part by veins. Here, and for a short distance beyond its borders, thermal and pain sense were entirely abolished while tactile sense was partly lost. Muscle and joint sense, arm, tendon and peri-osteal reflexes were good. Knee and ankle jerks were present and equal but not very active. The abdominal reflexes were good. The scrotal reflex on the right was markedly exaggerated, plantar reflexes were present and equal but not lively.

The two outstanding features of the case were the low level of the cord lesion and its limitation to one posterior horn.

Dr. Courtney suggested the following pathogenesis: The hikes probably acted as a trauma, producing hematomyelia. Then followed gliosis, then a breakdown of the gliotic tissue and cavity formation. Oppenheim states that he has seen only three such cases. With regard to the prognosis Dr. Courtney mentioned a woman seen a number of years ago, in whom the disease was well developed. He saw her again ten years later. Meantime she had borne three children. The disease had made no progress.

THE PATHOLOGY OF TRIGEMINAL NEURALGIA. DR. PERCIVAL BAILEY.

The pathogenesis of trigeminal neuralgia is still unknown. In the days of peripheral operations examinations of avulsed nerves were made, but the only suggestive finding was the so-called "beading" of the myelin sheaths reported by Drs. Mitchell and Spiller (*J. Nerv. & Ment. Dis.* **25**:400, 1898). Later on, when the ganglion was removed, various reports of examinations appeared. Of these, only four were concerned with nerves not previously subjected to operative interference. Head (Allbutt's System of Medicine, vol. 8, p. 724), Monari (*Brun's Beiträge* **17**:495, 1895), and Coenen (*Arch. f. klin. Chir.* **67**: 333, 1902) reported the ganglions they examined to be entirely normal. Schwab (*Ann. Surg.* **38**:696, 1920) thought there was some increase in interstitial tissue. Probably this was an error in interpretation, but even if correct, it is an unessential finding because no changes were found in the nervous tissues.

During the past year I have examined eleven ganglions from Dr. Harvey Cushing's operations, in only one of which, however, had there been no previous operative interference. For comparison sections were available from fourteen other cases in which there had been no previous operative measures.

In the one previously unoperated case, E. P., Surg. No. 14016, aged 50, the ganglion was immediately placed in 96 per cent. alcohol and stained with thionin. The specimen might have served as a model for one of von Lenhassik's plates. The ganglion cells were perfect. Nor could any pathologic changes be seen in any of the other structures. Sections from fourteen other ganglions previously unoperated showed no marked pathologic change. Three of them were fixed in liquor formaldehydi and stained by Marchi's method; three were fixed in liquor formaldehydi and stained with hematoxylin eosin, and eight were fixed in Zenker's solution and stained with hematoxylin and eosin or methylene blue-eosin. In the Marchi specimens considerable fat was revealed but not resembling that of secondary degeneration. For the most part, it consisted of the fat which normally increases in the connective tissues of the aged and the dirty deposit to be found in any nervous tissue which has stood in liquor formaldehydi. There was no "beading" of the myelin sheaths as described by

Spiller. The hematoxylin and eosin specimens were examined especially for any possible increase in interstitial tissue, and none was found. In the interstitial tissue of spinal ganglions, as well as in the trigeminal, were found numerous collections of closely grouped nuclei which resembled round-cell infiltrations. They had no pathologic significance. In some of these patients at operation the arachnoid may be seen to be thickened and adherent, but the same may be said of aged persons without trigeminal neuralgia. We may conclude, then, that the ganglions from patients with trigeminal neuralgia are essentially normal.

Eleven ganglions were examined from patients who had previously been subjected to peripheral procedures. In them also the pathologic changes found were exceedingly meager. One was studied by Marchi's method; two by the Marchi-Mallory combination of Jacob and the Marchi-Mann combination of Alzheimer; four by Alzheimer's method V on frozen sections; two by Nissl's method, and two by Bielschowsky's method in frozen sections and Hassin's Bielschowsky-Mann combination.

In neither of the Nissl preparations were there any signs of reaction at a distance. The ganglion cells not crushed by the scissors were well formed, filled with tigroid, the nuclei centrally situated, and there was no increase in fat or pigment content. The Marchi preparations showed rarely the "balls" of material which stain with osmic acid, as Spiller has described, but counterstaining, according to Jacob or Alzheimer, showed absolutely no reactive phenomena on the part of the Schwann cells. This finding cannot be accounted for. The material, whatever it is, lies within the myelin sheaths. There are absolutely none of the phenomena of secondary degeneration. Alzheimer-Mann and Bielschowsky preparations rarely showed a swollen or distorted axon, and never a fragmented one.

We may conclude that, aside from senile changes in the connective tissue and parenchyma alike and occasionally reaction at a distance in the cells from peripheral operations, the ganglions from cases of trigeminal neuralgia are normal. The essential lesion in this disease must lie in the periphery, probably in the nerve endings (Harris: *Brit. M. J.*, May 22, 1920). That section of the nerves at the peripheral foramina stops the pain until the nerve again regenerates is sufficient to establish this point, and after peripheral operations there is no reason to expect any changes in the ganglion other than the well-known minor ones in the cells, for the phenomena of secondary degeneration occur peripheral to the section and not central. So far as I know, the nerve endings have never been examined. Initiation of the pain by cold air, which causes reflex constriction of the vessels of the skin, and by talking, during which the skin is irritated by superficial muscles, etc., indicates a peripheral origin.

DISCUSSION

DR. HARVEY CUSHING said that the character of the pain, its unilateral situation and the manner of its spread, speak against the end organs as the seat of the lesion. Division of the nerves as well as alcoholic injections lead to certain changes in the ganglion which may be sufficient to inhibit for a time the discharge of paroxysms. It must be said, nevertheless, that trigeminal neuralgia is much more often bilateral than is commonly observed. About a year ago, all his gasserian patients were communicated with and replies were received from about 90 per cent. of the 360 patients. Possibly 4 or 5 per cent. of these patients after several years had begun to have pain on the other side. It is a curious thing that true neuralgia of this type proceeds by a definite

march. It goes from first to second to third or from second to third to first; it never jumps from first to third or the reverse, which would imply that the process lies in the center rather than in the peripheral nerve endings.

Dr. J. W. Courtney saw confirmation of Dr. Bailey's conclusion in certain clinical facts. In a considerable number of cases of migraine, certain attacks were characterized by pain limited strictly to the first and second divisions of a fifth nerve, with temporary residual tenderness on pressure of these divisions. From this he argued that as migraine results from a vasomotor ataxia, it was not unlikely that the pain in tic douloureux was the result of vasoconstriction of the nutrient arteries of the trigeminal nerve. This notion appeared to him all the more tenable because genuine tic douloureux most commonly occurs in arteriosclerotic patients.

Dr. Cushing replied that patients with major neuralgia were often young people, some actually in their teens, and he instanced a typical example in a lad of 16 years. He said that many of these patients had some disorder of the teeth or infection of the sinuses. Some of them, too, had a history suggestive of migraine, but trigeminal neuralgia rarely starts in the brow. Probably 60 per cent. of the cases start in the second division, 30 per cent. in the third division and only about 10 per cent. in the first division. Where there is primary supraorbital pain, one must be very suspicious of its nature.

Dr. Bailey said that the usual march of symptoms from one branch to another was more consistent with a peripheral origin, for if the disease were central, one might expect more often involvement of all three branches. The fact that one gets only a small part of the ganglion is offset by the fact that the part always removed contains the cells of the third division which is most frequently involved.

Dr. Ayer's remark might be answered by noting that section of the nerve peripheral to the foramina will not stop the pain in syphilitic basilar meningitis. The origin of the pain is not identical with that of trigeminal neuralgia.

LUMINAL POISONING. Dr. A. H. Ruggles.

In 1913, the dosage of luminal was given as from 1½ grains up to 12 grains. In *The Journal of the American Medical Association*, May 17, 1913, Dr. F. J. Farnell reported two cases showing toxic symptoms after the administration of luminal as a hypnotic. As far as is known there has appeared in American literature no similar report of the toxic effects of this drug; hence the report of this case.

E. G., a woman, aged 39, married, had always been somewhat nervous, tended to worry and could not stand fatigue or excitement. Married five years, she had no children, on medical advice. Following influenza a year and a half ago, she became more nervous and she had been in bed most of the time since. Her chief complaint was sleeplessness, and for the nine months previous to her admission to Butler Hospital she had taken 3 grains of luminal every night, with the exception of eight days when she tried to get on without it. During that time she is said to have slept brokenly or not at all. She took 1½ grains of luminal at bedtime and when she woke up during the night. On admission to the hospital speech was slow and slurring and the face expressionless. There was incoordination of arms and legs, difficulty in standing and a slow, unsteady gait. Sensation was normal. There was a tremor of the lips. The pupils were large but normal. Deep reflexes were normal. Luminal was at once discon-

tinued, but the disturbance of speech and gait continued for about two weeks. At the end of three weeks all the symptoms had disappeared, and the patient has recovered her former health.

This case seems to show that there is the possibility of toxic effects from the continued administration of luminal even in relatively small doses.

DISCUSSION

Dr. William F. Boos stated that luminal must be used with the greatest discretion, but if so used, it is very valuable. Its action is very much the same as that of other drugs of the barbituric acid group (barbital and derivatives). It is closely related to barbital, and the symptoms of poisoning are similar to those of barbital in poisoning, except that they are accentuated. The dose of luminal should never exceed 3 or at most 5 grains a day. A number of epileptic patients under treatment with luminal had never shown any toxic effects. At times there had been a slow, scanning speech, but that occurred when bromids were taken in addition to the luminal. The rash which is sometimes seen is brought on by the other preparations also. Dr. Boos had an interesting experience with barbital with a woman who was in the habit of getting very much intoxicated and who had lately been using perfume. She was given large doses of barbital, luminal, a little chloral and bromids. The next morning she awakened quite refreshed but complained of the doses being too homeopathic. During that day she drank more alcohol in various forms and became very much intoxicated. The next morning she sent to the drug store for a tube of ten barbital tablets. When seen not long after she had taken 35 grains in less than seven hours, and she showed symptoms like those of luminal poisoning. She appeared intoxicated, was unable to walk, spoke with difficulty, and her eyes had a peculiar dilated effect. Later she obtained three more barbital tablets, taking ten tablets in less than ten hours. The next morning apparently she was all right. Probably the after-effects of the alcohol acted as an antidote to the drug. Had she been a normal person, she might have been seriously ill, but as pain is an antidote to morphin, so the extreme nerve irritation that she suffered, the sleeplessness and restlessness and the sense of fear, were so potent that they succeeded in counteracting such a powerful drug as barbital. The effect on walking is marked in all these drugs.

The main point is that the action of luminal is very similar to that of all the derivatives of barbituric acid, except that it seems to be the most powerful.

A drug which can be strongly recommended is bromural. It is very much milder in its action and is practically harmless. It is also a derivative of urea. A young girl who attempted suicide took at one sitting more than fifty tablets. She went to sleep and slept for forty-eight hours, and then awakened refreshed. The correct dose is usually two 5-grain tablets every three hours.

Dr. Donald Gregg mentioned an epileptic patient who, under luminal treatment, became quite euphoric and pugnacious and much more troublesome than before. The epileptic attacks seemed to be abolished, but he became very hard to manage. There was no particular drowsiness or difficulty in walking.

Dr. Ruggles referred to a case of poisoning with this group of drugs in which the woman first took 90 grains of barbital sodium, then 110 grains of barbital and then 200 grains of barbital without any prolonged ill effects. There must be a good deal of individual idiosyncrasy in the reaction to these drugs, for there has been reported in the literature a case of death from 15 grains of barbital.

ANHEDONIA. Dr. A. Myerson.

A full abstract of this paper appeared in the July, 1921, issue of the Archives, page 101.

DISCUSSION

Dr. H. I. Gosline remarked that Dr. Myerson had taken certain symptoms from a number of diseases and had traced them back to certain psychologic categories. In introspective psychology the organic sensations form one of the fundamental groups. The next step would be to work back to the enteroceptive nerve fibers, the glands of internal secretion and all other anatomic entities capable of reaction and inhibition. Here is the beginning for a rational psychanalysis instead of an interpretative psychanalysis.

Dr. Myerson pointed out that the condition he had been describing was a syndrome. It may be traced back, as Dr. Gosline suggested, to a physiopsychologic and endocrine basis. All the psychoses may be traced to a physiologic basis. This condition occurs in many diseases, and it is the cardinal symptom in the manic depressive group. Whenever the energy content of the individual becomes disturbed, this condition tends to occur. It is a basic reaction of man to anything that interferes with his outward energy.

IS THE FACT OF A PSYCHOTIC PERSON BEING THE ELDEST OR YOUNGEST IN A FAMILY OF ETIOLOGIC IMPORTANCE? Dr. E. M. Pease.

A study was made of cases from the manic depressive and dementia praecox groups—forty from each, and an equal number from each sex in the two groups. These two psychoses were selected because they furnished 40.48 per cent. of all first admissions to the state hospitals of twelve different states during the year 1919. They also represent 25.80 per 100,000 of the general population.

As heredity forms one of the most important of the assigned predisposing causes of these two psychoses, a possible reason for this was assumed to be due in part, at least, either to immaturity of the gonadal plasm at the time of conception of the first child, or to the beginning of involutional regressive changes in the parent at the time of conception of the youngest child. A further suggestion is the possibility that the imbalance in the eldest child may be due to instability of the endocrine glands other than the gonads in parents of immature years or, in the case of the youngest child to atrophy or reduced function of the parental endocrine glands due to advancing years.

The group of cases studied was very small owing to incomplete data in many of the records consulted. To be more conclusive: The study should include the other psychoses, also a comparison with the corresponding group of families in which there have been no psychotic members. It is hoped that such data may be presented later.

The results of the study were: Of the whole series, 27.5 per cent. were the eldest and 18.75 per cent. the youngest children. The whole series also showed 30 per cent. of the males to be the eldest and 12.5 per cent. the youngest; of the females, 25 per cent. were the eldest and 25 per cent. the youngest. Grouped according to psychoses: In the manic depressive series, 32.5 per cent. were the eldest, and 15 per cent. the youngest children. Of these, 30 per cent. of the males were the eldest and 10 per cent. were the youngest, and of the females 35 per cent. were the eldest and 20 per cent. the youngest. In the dementia praecox series, 22.2 per cent. were the eldest and 22.2 per cent. were the

youngest. Distributed according to sex: 30 per cent. of males were the eldest and 15 per cent. the youngest, and 15 per cent. of females were the eldest and 30 per cent. the youngest.

CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, March 17, 1921

G. B. HASSIN, M.D., *President, Presiding.*

BEHAVIOR DIFFICULTIES. DR. DAVID M. LEVY.

DR. LEVY reported the case of a patient, aged 16, with dementia-praecox on whom mental tests were made over a period of two years.

Family History.—The mother had been very religious, the patient said that both parents had died as saints. She talked in a rambling, distractible way, bringing in useless details, but she managed the household well. The father's mother had had senile psychosis. The patient was the youngest of seven siblings; the other were employed chiefly as unskilled laborers. She had suffered from asphyxia at birth which required much effort in resuscitation. There had been no convulsions or fainting spells. She was a restless sleeper, but there was no sleep walking or enuresis.

Physical Examination.—This revealed an asymmetric skull and body, moist, cyanotic hands and feet, female distribution of pubic hair and moderate diffuse enlargement of the thyroid gland; pulse, 80; height, 65 inches; weight, 102 pounds; absent conjunctival reflex, sluggish corneae. Areas of hyperesthesia recorded in an examination made in 1919 were no longer present. Otherwise the examination was negative.

Mental Condition.—The patient received the Stanford revision of the Binet-Simon tests at the ages of 14, 15 and 16. In each case supplementary tests requiring briefer time intervals were also used (Trabue sentence completion) and were within a year of the Binet age. The mental ages scored were 13 years 4 months, 10 years 3 months and 8 years 6 months, respectively. This marked deterioration was in keeping with observations made by his school teacher, who spent much time with him because of his difficulty with simple problems in arithmetic which he had done with ease in the previous year. She observed especially that his difficulty with numbers was in marked contrast with his ability to read and his evident appreciation of difficult literary passages. In all these tests cooperation was secured, and the amount of deterioration determined (about five years) may be considered fairly accurate.

Of significance also is the fact that psychotic symptoms were not elicited until the third committment to the Detention Home at the age of 16. At that time the patient developed delusions of persecution, saying the doctors were all after him, they were trying to kill him; also that the world was going to end in three days; that he heard voices in which those words were announced. In the week previous to the expression of fairly definite delusions there was expression merely of vague fears and worries; "I'm worrying all the time, but it's about nothing in particular." The patient was committed on each occasion because of "incorrigibility" or stealing.

In the mental examination reaction time was slow, sometimes suspicious of blocking. He spoke with a slow, monotonous, depressed voice. He kept to himself in the ward. His dreams were filled with ghosts and spirits. When

he read he visualized the characters until he could see them all "as big as life" in front of him. "Brave by day, but quite a coward at night," said the mother, "trembling violently after he is told a ghost story."

Frequent masturbation had occurred with a few instances of homosexualism. Faulty attention was shown in his increasing forgetfulness when sent to the grocery on errands. His thefts, which were the cause of his commitment, always occurred when he was alone and on one occasion after worrying a great deal he returned a bicycle which he had stolen. He ran away, always alone. He never had "pals;" concerning the other boys, he said: "They always pick on me, call me names, always four to one against me."

At the time of the first examination the father's description of his behavior difficulties was: The boy was stubborn, impudent, tried to aggravate by constant argument, ran away when in difficulty; spent his money for playthings; if told to do something he would wait a long time and mockingly ask "What did you say?" (early negativism?). School progress was poor; he complained that his teachers were always assigning him too large lessons; he constantly used big words, "but he does not know what they mean," said the mother.

DISCUSSION

Dr. John Favill reviewed two cases of behavior difficulty diagnosed as psychopathic personality, egocentric type.

The first patient was a boy of 10 who stole, lied, smoked and chewed tobacco, was bad tempered, defiant, emotionally unstable and stubborn. Both parents were nervous. The home conditions were good except for an interfering grandmother. Physically there was a suspicion of mild hyperthyroidism. The intelligence quotient was 117.6.

The second patient was a boy of 10 who stole, lied, and was emotionally unstable and extremely stubborn. He had refused to confess his last theft for one week at home and a two hour interview at the Institute failed to bring out the truth. In the evening, however, he confessed to his parents. Heredity showed moderate nervousness in both parents and one older brother who stole for a while when a boy but who outgrew this habit. Home conditions were excellent. The physical findings were unimportant. The intelligence quotient was 139.1.

After obtaining the confidence of these boys and attempting by personal influence and suggestion to adjust their chief difficulties, good results have followed to date, in the first case for five months and in the second for three months.

These cases, selected from a large number of this type, some of which have been under observation and successful adjustment for much longer periods, appear to show the importance of personality factors over those of environment. They illustrate the fact that, with our modern understanding of the mental factors in behavior, the treatment by suggestion and by careful utilization of the circumstances in each case is sufficiently successful to encourage a wider application of this method.

INTELLIGENCE RATING IN THE PENAL INSTITUTIONS OF ILLINOIS. Dr. Herman M. Adler.

The results of the survery of the penitentiary at Joliet, briefly summarized, are:

The groups of native-born whites, foreign-born whites and negroes in the penitentiary rate somewhat higher in general intelligence than similar groups in the draft army. In each of these groups at the penitentiary there is a smaller percentage of men of inferior intelligence and a larger percentage of men of superior intelligence than was found in the same group in the army. Native-born whites in prison rate markedly higher as a group than foreign-born whites. Northern-born negroes rate higher than Southern-born. All whites as a group rate higher than negroes. There is little relationship between intelligence and the age of the prisoners.

Although all grades of intelligence are to be found among the different groups of criminals, there is a tendency among the groups to differ in general intelligence. Those in the fraud group possess the highest level of intelligence; this group is followed by the three groups, burglary, robbery and larceny; there is a low rating for murder, while sex crimes rate lowest in the scale of general intelligence.

There is a larger percentage of men of inferior mental ability in the groups of prisoners who have served five or more years at the penitentiary than among those who have served less than five years. Prisoners who have records of previous imprisonment as a group have a smaller percentage of men of inferior intelligence and a larger percentage of men of superior intelligence than the group of prisoners against whom there is no such record. There seems, therefore, to be a general tendency for recidivists to rate somewhat higher in intelligence than first offenders.

Of the group of men of inferior intelligence by far the great majority are negroes and foreign-born. Less than two-tenths of one per cent. of the entire prison population may be classified as imbecile according to their intelligence ratings. Sixteen per cent. of the population rated below 11 years mental age.

There is a close agreement betwen the results of the survey of the penitentiary at Joliet and the reformatory at Pontiac. The population of these two institutions represents the majority of the adult offenders in the state of Illinois whose crimes constitute the more serious offenses. The intelligence surveys of these institutions do not indicate that there is a close relationship between mental deficiency and delinquency among this class of delinquents.

DISCUSSION

DR. CLARENCE NEYMANN, at the psychopathic hospital, was struck by the fact that those people who were so prone to get into difficulties with their surroundings and made the greatest amount of trouble were usually people of average intelligence and were not, strictly speaking, patients who were mentally defective. Such patients were usually brought in by the social service departments with the report that the patient spent his money in getting drunk or for morphin and did not support his family—perhaps supported another woman. Dr. Neymann always doubted the strict correlation of feebleminded-ness and insanity with behavior difficulties. Some people are troublesome to the community on account of mental deficiency, but most of them are not feebleminded. People with psychopathic personalities are unusually quick to adapt themselves to institutions and always arouse the sympathy of the physicians, especially of the interns. They always tell a good story, promise never to repeat their offense, but on going out they do the very things they have been warned against, and are brought back to the institution—only to make the same promises again.

The drug cases, the chronic alcoholics, the men who do not support their families, the men who commit sex crimes or peculiar sexual acts, the men at war with their environment—these patients are usually neither insane nor feebleminded, but have psychopathic personalities. This point should be recognized, and one should not attempt to solve all behavior problems along the lines of feeblemindedness and insanity.

Dr. Julius Grinker agreed with Dr. Neymann that there is a conspicuous absence of so-called dementia praecox in the penal institutions. He compared the statements of Dr. Adler with those made by others that almost every juvenile delinquent brought into court has a case of dementia praecox.

Dr. Grinker thought it was gratifying to hear some one who had made a profound study of the subject honestly admit that studies have just been begun and that definite conclusions cannot be drawn from so few figures. Such studies as Dr. Adler is making must ultimately lead to definite conclusions and meanwhile have a tendency to stimulate further research.

Dr. Herman M. Adler (closing the discussion) said the answer to Dr. Reitman's question depended somewhat on the personalities involved in the situation. In Illinois the staff officers of the institutions have no administrative authority. Such authority was refused because the success of the officers depends on their not having such authority. They have no power to order a patient in or out of hospital, they cannot order "punishment" or "no punishment," but act simply in an advisory capacity. In the penitentiaries they have an advantage over the physician in the state hospital in that in the state hospital there is a staff of physicians, and the head of the hospital is a physician who knows more than any one else on the staff. An assistant makes a recommendation, but the superintendent *is the physician* and can carry it out or not, as he wishes. In the penitentiaries they are all lay officials, and the warden has full charge and can do anything he wishes. If the psychiatrist tells him a certain prisoner is demented and should not be punished the warden can let him go unpunished or not, as he wishes, but if the psychiatrist makes correct diagnoses and does his work well, the warden will not take the chance of disregarding the advice, especially if the physician makes his diagnoses in writing and sticks to them. There will not be many trials before his word is carried out. Dr. Adler thought it was encouraging to know that the state penitentiary at Joliet, which was known as one of the worst in the country so far as structure and habits are concerned, has done better than any other of the state institutions. There the staff actually amounts to something and it passes on the problems suggested. This has been brought about by the great decency of the institution as a whole and the fact that the psychiatric work has been done carefully and properly and no recommendations have been offered that were not justified. The warden has turned over practically all the staff work to the psychiatrist and the old-time guards, that they had been warned against, come to the psychiatrist and put their troubles before him and seek his advice in the handling of many of the prisoners. If a man does not obey promptly and adapt himself to all the rules quickly they think there is something wrong with him and ask the psychiatrist to look him over—which sometimes proves embarrassing when the psychiatrist cannot find anything wrong. Instead of having to force their services on the staff they are really a little "over sold," and are trying to hold back and find out the answers to some of the problems. The same holds true of conditions at Pontiac and St. Charles and Geneva.

Dr. Adler thought the crux of the matter is that they did well in diagnosing and classifying people, but when it comes to making good on some of the promises about treatment and prevention they are a little short. In his opinion it is time to stop some of the propaganda and do some constructive work.

PHILADELPHIA NEUROLOGICAL SOCIETY

March 25, 1921

GEORGE WILSON, M.D., *President*

A CASE IN WHICH SEVERE GENERAL ATAXIA DEVELOPED DURING THE COURSE OF ACUTE ENCEPHALITIS. DR. WILLIAM B. CADWALADER.

This case is presented in order to call attention to the tendency to recovery in a type of severe generalized ataxia that may develop in acute encephalitis occurring in children. In this case the type of encephalitis could not be determined. Although severe ataxia may continue long after the acute symptoms have subsided, there is a tendency to improvement, in some instances with complete recovery.

History.—C. B., 3 years old, was admitted to the Presbyterian Hospital, Nov. 7, 1919. He had been healthy until two days before admission, when he was suddenly seized with abdominal pain, severe occipital headache and vomiting. The following day he had a series of general convulsions in which the lower limbs were held strongly flexed and the upper limbs were rigidly extended.

Physical Examination.—The pupils were equal and the eyeballs moved normally, but there was moderate lateral nystagmus. Moderate rigidity of the neck was noted. The cranial nerves, thoracic and abdominal organs and the extremities seemed to be normal, though there was a tendency to flaccidity. The tendon reflexes were absent. On admission the temperature was 102 F., the pulse was 132 and the respirations were 22. Blood culture and blood count were negative. The spinal fluid was clear and contained 7 mononuclear cells per cubic millimeter. One week later there were 19 mononuclear cells, and on two other occasions 7 and 10 cells.

Course.—A diagnosis of meningo-encephalitis was made. On November 14, one week after onset, there was moderate incoordination of the upper extremities with marked flaccidity. The fever continued, varying from 99 to 101 F. The incoordination gradually increased and involved the muscles of the neck, trunk and limbs.

The general symptoms did not subside until March 31, 1920. At that time the incoordination was so general and so severe that the patient was unable to feed himself, unable to stand, or even sit up in bed without support. With every voluntary movement the limb was thrown about in a wildly ataxic manner. When at rest there was no tremor. The limbs were extremely hypotonic, but not paralyzed. Hearing was impaired and there seemed to be some mental deterioration.

At present the patient is still moderately ataxic, but can walk although he frequently falls. His mother states that he can go up and down stairs. The tendon reflexes are absent, vision is good and there is no longer nystagmus. The incoordination or ataxia seems to be progressively diminishing.

TWO CASES OF FAMILY SPASTIC PARAPLEGIA. Dr. N. W. Winkelman.

The mother of the patients is in good health but has external strabismus. The father has had tinnitus for fourteen years with neither dizziness nor deafness. There are four boys and three girls in the family. One boy died at the age of 1 year from an unknown cause. The elder of the patients, an 18-year old girl, born in Russia, was admitted to the University Hospital, Nov. 23, 1920, because of tremor and difficulty in walking. Three years ago it was noted that the patient had tremor of the hands, and a year later she began to walk on her toes with spastic gait. The trouble has gradually increased so that now she has much difficulty in walking. She has received six injections of arsphenamin but has shown no improvement. She was born by a breech presentation and was asphyxiated. She had the ordinary diseases of childhood.

Examination revealed a mentality decidedly below par. The pupils were equal, regular and reacted normally. There was weakness of the right external rectus muscle, but no nystagmus. The reflexes of the upper extremities were increased but equal. Slight spasticity of the upper extremities was present. Rapid movements were awkwardly performed. The knee jerks were much exaggerated, the right being greater than the left. There was no ankle clonus, but the Babinski reflex was present on the right with a suggestion of one on the left. The legs were stiff. No impairment of sensation was found and no atrophy. The laboratory findings were: albumin and hyaline casts in the urine, blood count normal, Wassermann reaction and spinal fluid negative.

The other patient, the 2-year old brother, had a normal birth. At the age of 5 months the child fell out of a coach and since that time had been cross-eyed. While the child was still very young the mother noted a little difficulty in spreading the child's legs. He began walking at 20 months but has never walked normally. He is unable to talk. For the first time, a month ago, he had seven spells in one day but has had none since.

Examination revealed a child backward mentally, with internal strabismus. The upper extremities were normal. There was slight spasticity of the adductors and less of other muscles of the legs with a slight tendency to drag the legs. The reflexes were increased but no ankle clonus or Babinski sign was present.

A CASE OF DECEREBRATE RIGIDITY. Dr. J. W. McConnell.

R. C., a boy of 5½ months, was born after an easy labor. Apparently the child was normal at birth, except for distortion of the head which quickly disappeared. In less than a month after birth unusual stiffness of the whole body and a tendency to what was called "shivering" was noticed. When awake the child was constantly crying or moaning as though in pain, but nursing or a bottle of water would quiet him for a short time.

He was brought to the dispensary at the University of Pennsylvania, March 17, 1921, at which time the notable features were extreme opisthotonos and stiffness of the whole body.

Examination revealed a well nourished child. Eye movements were normal; there was no nystagmus; the pupils were normal. The head was of normal size, the anterior fontanel was unusually large; the posterior one more easily determined than it should be. There was no paralysis. The striking feature

was hypertonicity of the entire body. Legs and arms were held very stiffly. The arms were flexed at the elbow, overextended at the wrist, with fingers flexed tightly over the thumbs. There was extreme opisthotonos; the head retracted between the shoulders; the legs were in complete extension with overextension of the feet so that in the opisthotonic position the supports of the body were the head at about the posterior fontanel and the soles of the toes. Despite this extreme hypertonicity the limbs were frequently in tonic-

Fig. 1.—A case of decerebrate rigidity showing opisthotonos, retraction of the head, spasticity of the limbs, extreme pronation of the hands and extreme hyperextension of the feet.

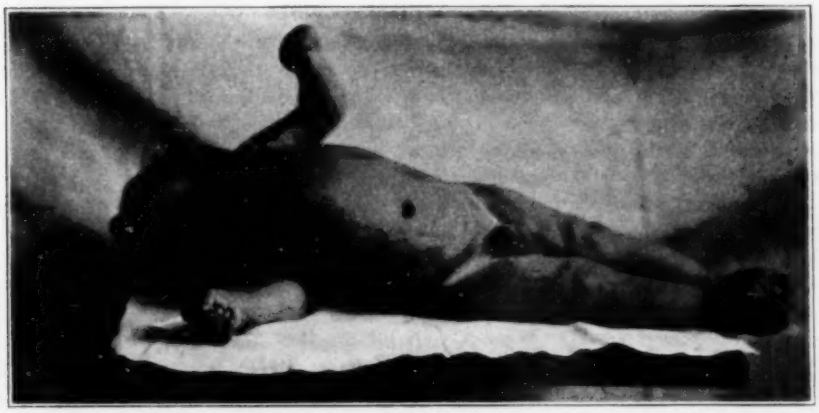

Fig. 2.—Case of decerebrate rigidity. The child is lying on his right side.

clonic movement, intermittent as to occurrence, and affecting the limbs as a whole so that they had the appearance of being shaken in the air with the shoulder joint and hip joint as the points of fixation. During all this time the child seemed to be quite conscious and was crying or moaning, the sounds being synchronous with expiration. The respirations were jerky and irregular with sighing once to every ten respiratory movements. The preferred position was on one side, usually the right. When placed on the back the opisthotonos interfered with the child's ease, and almost immediately the body would

fall to the right side, rarely to the left. The hypertonicity was uninfluenced by position. Occasionally after gentle friction there was slight temporary relaxation. The intermittent tonic-clonic movements seemed to be uninfluenced by position or handling. A tentative diagnosis was made of decerebrate rigidity, with tonic so-called cerebellar type of convulsions.

DISCUSSION

Dr. W. G. Spiller said that the child presented a typical picture of decerebrate rigidity; the extension of the limbs and pronation of the hands were typical, but flexion of the toes was lacking. The condition was what Kinnier Wilson had described as decerebrate rigidity. Wilson called attention to the pronation of the hand in chorea and athetosis, and it was striking that many disorders presented more or less a picture of this condition. It was astonishing that this syndrome had waited so long for a description by Wilson or someone else, and it was noteworthy that Wilson had his first case in 1905 and did not report it until last year, and yet the clinical picture was so striking that no one could fail to recognize it.

Dr. J. Hendrie Lloyd asked why this case could not be an instance of posterior basilar meningitis, such as Barlow described and whether a spinal puncture had been made.

MICROSCOPIC SECTIONS FROM A CASE OF SYPHILIS OF THE EIGHTH NERVE. Dr. J. H. Lloyd.

This presentation was incorporated in a paper published in the May number of the Archives of Neurology and Psychiatry, pp. 572-579.

DISCUSSION

Dr. Charles K. Mills, referring to the possibility of the arsenical treatment causing deafness, reported the case of a boy about 15 years of age, who had acquired syphilis and who had, within a few months, developed symptoms of eighth nerve involvement and had become deaf rapidly. He, of course, was treated by the old fashioned method with full doses of mercury and iodid and yet went on to complete and irreparable deafness.

Dr. Francis X. Dercum believed Dr. Lloyd's case was one of syphilis of the eighth nerve. If deafness is caused by arsphenamin it is remarkable, considering the enormous use of the remedy, that deafness does not occur more frequently. Deafness in nervous syphilis occurs every now and then when arsphenamin has not been used. Such a case from Dr. Dercum's clinic was presented by Dr. Gilpin before the Neurological Society a few months ago. The man's condition subsequently decidedly improved as a result of arsphenamin treatment.

Dr. N. W. Winkelman said that they had not stained for spirochetes because the stain would stain axis cylinders, and if they were broken up, it was difficult to distinguish the spirochete from the axis cylinders.

Dr. H. S. Newcomer reported a patient, about 16 years of age, who received arsphenamin at irregular intervals from the time he developed secondary infection. After he had been under treatment for four or five months he developed deafness and dizziness, for which he was given weekly arsphenamin treatments and simultaneous intraspinal injections of mercurialized serum. At the end of about four weeks he had recovered from the deafness. There was a recurrence of dizziness but not of deafness which was similarly treated. For several years he has had no further symptoms.

Dr. J. Hendrie Lloyd said that others had observed cases like that of Dr. Newcomer, whose patient had developed deafness in the secondary period, and who had recovered from this deafness when the treatment was pushed. Dr. MacKenzie of Philadelphia, who had written the best paper on syphilitic deafness that Dr. Lloyd had ever read, said that they were not cases of arsenical poisoning; that a sufficient quantity of the drug had not been given. Dr. Lloyd said he had seen three cases of deafness develop rapidly after the administration of arsphenamin. The loss of hearing came on in a few days and was hopeless. He thought the cause was syphilis, but whether arsenic had something to do with it he did not know.

A CASE OF FRIEDREICH'S ATAXIA: EXHIBITION OF MICROSCOPIC SECTIONS. Drs. J. H. Lloyd and H. S. Newcomer.

This paper is published in full in this issue, p. 157.

DISCUSSION

Dr. N. W. Winkelman said that he had been very much interested in the sections. His first thought was that they resembled early tabes. The stained sections looked exactly like the drawings made by Trepinski years ago when he worked out the myelinization of the posterior columns. In the sections he saw the roots were normal. There was no doubt that in the lower cord the pyramidal tracts were affected and the cerebellar tracts higher up. Dr. Winkelman saw one section of cerebellar cortex which he thought was very much narrower. He did not think the granular layer had as many cells as a normal cerebellum. The Purkinje cells apparently were normal in number, but much decreased in size. They stained well. He would not have said that the cerebellum was normal.

Dr. W. G. Spiller said that the importance of this case was in the youth of the patient. The findings were valuable in showing the early lesions of Friedreich's ataxia. They brought up an important question: Are we to assume from the resemblance of the degeneration of the posterior columns to a certain stage of fetal medullation of these columns and to the degeneration of early tabes that this degeneration of the posterior columns in Friedreich's ataxia is exogenous in origin, i. e., that it begins outside of the cord? Dr. Spiller said that he had shown some years ago that many naked axis cylinders persist in the posterior columns, and by this fact he explained the preservation of sensation. The posterior columns in Dr. Lloyd's case showed typically the tourbillon described by Déjerine and Letulle, which had caused considerable discussion. He thought that one must be extremely careful in judging degeneration of Clarke's columns. The cells in these columns show changes more readily than almost any other cells of the spinal cord. Sections can be made through various parts of the thoracic region in normal cords without showing many of these cells. The region of greatest development of the columns, about the second lumbar segment, should be studied before one gives an opinion as to the scarcity of these cells.

Dr. J. Hendrie Lloyd said that the examinations showed that the cerebellum was normal, but he did not know whether the cerebellar peduncle had yet been examined. The large cells of Clarke's columns were destroyed at many levels. The inferior cerebellar peduncle consisted of six or seven sets of fibers; they were the connections with the cerebellum which one would think would be impaired. The cerebellum itself was not involved according to the

opinions of all who had seen the sections. This cord was not syphilitic. The lesion was more than a posterior sclerosis. It was customary to speak of Friedreich's ataxia as a posterior sclerosis, although it is known that it is more than that. What the exact process was, Dr. Lloyd thought remained unsolved. It was a disease *sui generis*, of unknown causation.

Dr. H. S. Newcomer said he could not say what level of Clarke's column was examined. Six or more levels in the dorsal cord were examined. Most of them showed very few Clarke's cells and those which were present showed a great deal of sclerosis and simple chromatolysis. The tract sclerosis in the posterior columns was not of the tabetic type in which the fibers are laid down parallel to each other (columnar arrangement), the field being stippled with the ends of the fibers. The sclerosis here consisted of an irregular and interwoven mat of glia fibers.

SAN FRANCISCO NEUROLOGICAL SOCIETY

Regular Meeting, June 3, 1921

Milton B. Lennon, M.D., *President, in the Chair*

A CASE OF MYOTONIA ATROPHICA WITH ASSOCIATED BRADYCARDIA. Dr. M. H. Hirschfeld.

In addition to the classical findings in Thomsen's disease, this patient presented atrophy of the shoulder girdle muscles and periods of bradycardia.

History.—W. H., a young man, aged 20, whose family history was not known except that one sister had had a similar condition which had improved slightly, had attended school and had done general labôring. His habits and past history were negative. For as long a time as he could remember he had had his present difficulty in initiating movements. He felt muscle bound, but after performing a series of muscular movements he was able to proceed normally. Sudden fright or slight interference with his activities might throw his entire musculature into spasm and result in a fall. The malady involved the orbularis palpebrarae and muscles of mastication, but speech was not affected.

Examination.—He had a myopathic type of face. The shoulder girdle was atrophic, particularly the deltoids and rhomboids. Pectoral and biceps muscles were very small; they were unusually well developed in childhood. He had a wasp waist and lumbar lordosis, hypertrophied calves, quadriceps and glutei and active tendon reflexes.

All the body musculature, both hypertrophied and atrophic, showed the typical myotonic reflexes described by Erb, with the exception of the wave formation with stabile galvanic current, up to 20 milliamperes. However, slight variations in current strength produced contractions.

The basal metabolism, Wassermann reaction, lumbar puncture, blood sugar, and blood and urine creatin and creatinin showed no abnormality.

Course of Illness.—The day following the lumbar puncture bradycardia (40 beats per minute) developed and continued for one week. Under atropin the rate rose to 64. Electric cardiograms showed slowing of the entire cardiac cycle, particularly marked during ventricular systole. The patient gave a history of similar attacks with no known cause. Sections from the atrophic deltoid muscle and the hypertrophied glutei and vasti were normal.

DIFFERENTIAL PUPILLOSCOPE OF C. VON HESS. Dr. Otto Barkan.

With this instrument the intensity of light, which just suffices to cause the pupil to react, is accurately measured. This is read off on a scale and the figure used to express the "motor discriminative acuity" of the case in question. In normal cases a difference of from 95 to 100 suffices to cause pupil reaction. Comparison of the numerical values of the direct and consensual reactions, with other data, greatly assists in localization of the lesion in the reflex arc, even in the presence of optic atrophy or cataract. The method has proved of particular value in the diagnosis of incipient tabes, as by means of it a pupillary lesion can be definitely diagnosed long before any change in the pupil reaction can be detected with ordinary methods.

Book Reviews

PSYCHOLOGY AND PSYCHOTHERAPY. By WILLIAM BROWN, M.A., M.D. (OXON.), D.Sc. (LOND.), Reader in Psychology in the University of London (King's College), Clinical Assistant in Neurology, King's College Hospital, Late Medical Officer in Charge of Craiglockhart War Hospital for Neurasthenic Officers, Author of "The Essentials of Mental Measurement"; with a Foreword by WILLIAM ALDREN TURNER, C.B., M.D. Pp. 196. New York: Longmans, Green & Co.; London: Edward Arnold, 1921.

The bulldog is slow but tenacious. Probably England was the last civilized land to take a serious look at the psychoneuroses, and the war roughly taught her that she knew little of them. Having, with becoming deliberation, learned the lesson, she has now developed quite a crop of neuropsychiatrists or dynamic psychologists, and it would not be surprising a little later to find there the most uncompromising and dogmatic followers of Freud or Jung or Janet or Dubois, or a group of equally rigid psychopathologists of a distinctive English school.

The book of Dr. Brown, who is one of the younger English neurologists, is essentially a war product written by an honest, educated, thoughtful man who had a rich experience both just back of the front and in a neurologic service in England. Consequently it is a good book. But like many other authors who were decidedly immature when their war experience began and consequently had to take in a huge mass of information and experience in a too intensive fashion, Dr. Brown shows some evidence of indigestion and imperfect assimilation. Evidently he has tried to be broad-minded and catholic. Here and there he accepts more or less of the ideas of Charcot, Janet, Freud, Jung, Fierenczi, Dubois, Déjerine, Babinski, Prince, McDougal, Bergson and others. Indeed, as a composite picture—a bit blurred—of present day views of the psychoneuroses, the book will be interesting to all and decidedly instructive for the beginner, be he a young or old practitioner. Only two things are lacking; both can be corrected by time and experience. First, there is lack of self determination by the author. Except as regards dissociation and abreaction, he is not quite sure of himself. He grants too much to be entirely consistent. Second, the lesson of the war neuroses is not carried over into civil life.

The book is made up of five parts. The first part (two chapters, 35 pages) is introductory and briefly discusses the psychopathology of dissociation, multiple personality, psychocatharsis, the subconscious and the like. The second part (three chapters, 61 pages) is principally devoted to Freud's theories. The author distinctly disavows being a Freudian, but gives great weight to psychanalysis, 45 per cent. of the text being devoted to this subject. Part 3 is headed "Psychotherapy" (20 pages), but consists wholly of theoretical consideration of various factors operative in different methods. Part 4 (three chapters, 50 pages) is a discussion of the psychoneuroses of war, their nature, varieties and treatment. Chapter 9 is a useful chapter in which are compared cases of war neuroses seen early and those seen later. Just why the author should have tacked on Part 5 (a chapter of 22 pages) is not apparent. It is a rather

discursive, somewhat immature and wholly useless disquisition on the relation of the mind to the brain, in which he finally is unwilling "to cast any doubt on the results of scientific investigation into spiritism."

The strong points of the work are the author's honesty with himself and the reader, his earnest attempt to get at the nature of the psychoneuroses and their best treatment, and his clarity of style.

The reviewer once heard an American jokingly assert to an English friend that in England crystallization is hereditary and congenital. As regards this little book of Dr. Brown, nothing could be farther from the truth. It is plastic almost to the point of confluence. Whether or no the reader accepts the author's conclusions, he has a fair and good presentation of the subject, a reasonable basis for opinions of his own.

The author is an implicit believer in abreaction or psychocatharsis in the treatment of hysteria and uses hypnotism to bring it about. Incidentally, it is interesting to note that many English neurologists have had to go through the evolutionary stage of hypnotism so long ago passed by other nationalities. Some of Dr. Brown's cases of abreaction in hypnosis are striking, and apparently his conviction of its scientific soundness is based on results of treatment—a rather shaky foundation when dealing with hysteria. Another interesting item is a collection of the histories of twenty-two patients treated in the field whose after-histories were obtained. All were treated with light hypnosis; only one had a relapse and fifteen returned to duty.

For long standing, intractable cases the author believes in what he calls autognosis. That is, by means of repeated explanatory, educative, persuasive talks the patient is given insight into his condition. Two cases are given in detail.

Perhaps partly because the book is to a considerable extent made up of discrete articles already published, it contains some careless statements and some contradictions. One or two of these may be mentioned. The unqualified statement is made that "hysterical patients are easily hypnotized." Of course, many of them are. Possibly every one of them could be hypnotized if all conditions, including the operator, were right, but certainly no one physician can hypnotize all hysterical patients.

On page 10 the author says that hysteria is "due to an emotional shock," whereas it probably never is. There are always preparatory mental processes, or there is mental elaboration following the shock. Indeed, the author himself later makes this plain.

Speaking of a (blindfolded) hysterical patient who said "yes" each time a normal area was touched and "no" each time an anesthetic area was touched, the author says "There is dissociated from the *entire mind* (italics ours) one psycho-physical power, in this particular case cutaneous sensitivity."

A pitifully small bibliography and a good index conclude the volume.

THE OXFORD MEDICINE. By Various Authors; Edited by HENRY A. CHRISTIAN, A.M., M.D., Hersey Professor of the Theory and Practice of Physic, Harvard University; Physician-in-Chief to the Peter Bent Brigham Hospital, Boston; and SIR JAMES MACKENZIE, M.D., F.R.C.P., LL.D., F. R. S., Consulting Physician to the London Hospital, and Director of the Clinical Institute, St. Andrew, Scotland. In Six Volumes, Illustrated. Volume 3, Diseases of the Digestive System, Kidneys and Ductless Glands. Pp. 828. New York: Oxford University Press.

This volume discusses diseases of the gastro-intestinal tract, the liver and pancreas, the kidneys and the ductless glands, except the thyroid; an article on this gland, by Plummer, is to appear later. It is a pleasure to be able to

say that this volume maintains the standard set by the second volume. While a carping critic might point out some typographical errors and some curious English, such for example as a "reasonable reason," omitting the customary "if you know what I mean," these are not of sufficient moment to lessen the value of the articles.

As might be expected, the first article is on diseases of the esophagus, by Frothingham. The article is quite conventional and occupies a space proportionate to the importance of these diseases, but one might ask why, in a brief chapter, space is given to diseases of no clinical interest that might far better be given to the few important diseases of the esophagus.

Rehfus follows with a discussion of diseases of the stomach, excluding gastric ulcer. With proper force he points out the frequency with which gastric symptoms appear in a wide variety of diseases, a fact frequently forgotten not only by the general practitioner but also by the gastro-enterologist: "It behooves the physician therefore to treat every gastric case as one which is fraught with not merely possibilities of gastric disease and to recognize that even more frequently gastric disease is the expression of some systemic fault." Bad English, but an important clinical fact.

The discussion of the physiology of the stomach and of the methods of clinical examination is adequate.

It would be well for many general practitioners to read with especial care the chapter on gastro-enteroptosis, that they may avoid making this diagnosis as often as they do. Some of our surgically inclined friends could do the same with considerable profit.

Gastric carcinoma and gastric syphilis are well handled, but it seems to the reviewer that the author has given less discussion to functional disturbances than their frequency would warrant. We believe that gastro-enterologists are too much inclined to neglect cases of this type, or more properly speaking, to look on them as organic diseases. They overlook the patient in their interest in the examination of his gastro-intestinal tract.

In the chapter on ulcer of the stomach and the duodenum, Sippy again expresses his conviction as to the supreme value of alkalies in the treatment of these cases, and apparently his assurance increases as his already large experience grows. He leaves one in doubt as to his opinion on the importance of focal infections in the causation of ulcers and in their resistance to healing, but mentions in the paragraphs on treatment that foci of infections should be sought and removed.

His plan of treatment remains essentially unchanged and his indications for surgical treatment should be carefully read by all surgeons, especially by those whose writings suggest that any one who does not operate immediately after making a diagnosis of gastric or duodenal ulcer approaches the class of the criminal moron.

Stockton has written a carefully prepared, possibly too long, article on diseases of the intestines, with what appears to us to be marked disproportions in the distribution of space. This however is a matter in part of personal opinion, and he could no doubt justify giving nearly as much time to a discussion of the interesting but infrequent Hirschsprung's disease as to the common and troublesome chronic colitis.

Rolleston's articles on diseases of the liver and biliary tracts, illustrate the fact that generally the English are more careful in their writing than are Americans. While style, no doubt, is less important than material, a fact well

stated makes more impression and is pleasanter to read than one badly stated. His discussion of the various types of jaundice is satisfactory, containing clear statements on a matter about which there is much confusion of thought.

Christian's chapters on nephritis are worthy of especial praise. They are clear and clean cut and gain much by his use of illustrative cases. The modern methods of testing renal function are adequately considered, and his classification of nephritis is satisfactory because it is clearly only a working basis, easily subject to such revision as must come with increasing knowledge. It is a relief to see a classification not based on pure speculation and not including fifty-seven varieties. This is a thoroughly good article.

The chapters on the suprarenals and especially on the pituitary gland are too sketchy and appear to have been abridged at the last moment.

ORTHOPEDIC SURGERY OF INJURIES. By VARIOUS AUTHORS. Edited by SIR ROBERT JONES, K.B.E., C.B., F.R.C.S., Director of Orthopedics, St. Thomas' Hospital, Surgeon, Royal National Orthopedic Hospital, Consulting Orthopedic Surgeon, Royal Infirmary, Liverpool, Honorary Adviser to the Ministry of Pensions (Orthopedic Surgery). Two volumes. Pp. 512 and 657. London: Henry Frowde, Hodder & Stoughton, 1921.

Volume 1 is devoted to deformities due to injury and gives one the distinct impression that it was intended to be a military manual. The material is almost entirely from military sources, and two full chapters are devoted to a discussion of team work between the casualty clearing station and the base hospital. Another is entitled "Splinting of War Fractures," and another is given over to the work done in centers for the limbless.

There is an excellent chapter on "Orthopedic Surgery of the Hand and Wrist" by Walter I. Baldwin. This contains much that is of great value in reducing the disability from this class of injury. A chapter by Sir Robert Jones on the relation of deformity to ankylosis and stiff joints is valuable. He gives clear instructions as to the positions of choice, as to breaking up adhesions, when to interfere and when not to, and gives his reasons for his beliefs.

The chapters on fractures, malunion and ununited fractures add nothing to recent developments along these lines. The book is an exposition of the methods of treating war injuries. It contains much valuable material which can be adjusted to the treatment of injuries in civil life.

The greater part of volume 2 is devoted to consideration of injuries of the central and peripheral nervous systems. Injuries of the head and of the spine are discussed by E. Farquhar Buzzard and Percy Sargent. The subject matter is good, but the space devoted to these subjects is so limited that one who is neither a neurologist nor a neurosurgeon will be little benefited by these chapters.

The anatomy of the peripheral nerves, to which the first chapter is devoted, is adequately described by A. Melville Paterson—obviously an abridgement of his monograph on the same subject. Many of the illustrations have been borrowed from standard textbooks on anatomy.

The chapter on diagnosis of injuries of the peripheral nerves by T. Granger Stewart and W. Rowley Bristow is well illustrated and fairly comprehensive. One useless feature is the inclusion word for word of the anatomic description of each of the nerves as it has already appeared in chapter 1 under anatomy of the peripheral nerves. This is mere padding and uses space which could be utilized for fuller clinical description.

An exceedingly well written chapter on the operative treatment of war injuries of the peripheral spinal nerves is presented by Sir Harold J. Stiles. It deals adequately with the surgical anatomy of the peripheral nerves and is very well illustrated.

There are two chapters on the end-results of nerve injuries and operations. These are valuable, but the space occupied by the tables does not appear to be justified by the lesson they teach.

A chapter on purely functional and reflex disabilities in their relation to orthopedic surgery is presented by L. leF. Burrow and William Cuthbert Morton. Some of the statements in this chapter probably will receive criticism at the hands of neurologists. For example, "orthopedic cases suffering from purely functional disabilities are seldom hysterical" and "there is reason to believe that any muscle which is ignored or suppressed may in time pass beyond the control of the will, possibly because it has become anesthetic through disuse." Again under treatment they state that "no patient is ever treated as a hysteric or malingerer."

"Trick movements" in cases of nerve lesions are very well described by Fredrick Wood Jones, and by means of double exposure photographs well illustrated.

The continuity of the material could, perhaps, be improved by placing the subjects dealing with diagnosis of peripheral nerve lesions, such as trick movements, next to the chapter on the clinical consideration of injuries to the peripheral nerves and placing the chapters on injuries of the head and spine after the chapters on peripheral nerves.

With few exceptions, the material included in this volume represents careful and accurate study of the types of injuries observed in the late war.

THOUGHTS OF A PSYCHIATRIST ON THE WAR AND AFTER.

WILLIAM A. WHITE, M.D. Pp. 137. New York: Paul B. Hoeber.

Dr. White, whose competence in the field of constructive psychiatry is known through his "Principles of Mental Hygiene" and "Mechanisms of Character Formations," has presented in this small compass the important comments of the psychiatrist both on the psychology that leads to war, and on the conditions which war superinduces and leaves behind. He presents the individual as a bundle of instincts that seek his own preservation and that of the species. He shows him existing, however, in a society necessary to his own life and training him to its own larger purposes. The process by which the organization of the individual into the social organism takes place is through repression of the individual instincts, so far as these run counter to the social functions, and their sublimation into socially valuable activities. Suppression takes place through the "herd critique," sublimation through "the utilization of the energies so repressed to find satisfaction in ways that are progressively more and more removed from primitive types." "The force that deflects the primitive instincts of love and hate from immediate satisfaction . . . is that group of necessities which arise in consequence of man's living together in groups, the so-called instinct of gregariousness, or herd instinct. "To put it another way: The force which, in its negative aspect makes for repression—the herd critique—produces in its positive aspect the desire for the reward of social esteem. As individuals are organized into groups, so are groups and communities organized into larger communities, nations and international groups. This takes place by a process of differentiation of func-

tion and organ. The process of this differentiation is away from primitive instincts and their immediate expression. Let the organization break down, and the primitive 'infantile' instinct reaches expression. Thus wars arise in the breakdown of international organization, and provide the opportunity for rebuilding, 'rejuvenesce,' an opportunity which has been necessary in the past. It remains to be seen whether a community of nations can be built up in the future which will be able to grow without this process of destruction precedent to rebuilding." The principle theme of the book is the display of primitive instincts, as the nature of the individual, and relatively unconscious, over against the social nature which has been subject to the training of the group. With any breakdown in this organization appear the primitive instincts, in what may be called their infantile form. This unconscious group of instincts the author considers selfish, and their appearance is in varying degrees, abnormal and degenerative. With full recognition of the opportunities which war presents for the self-sacrificing impulses and the higher ideals, Dr. White shows that in large degree war must, with its breakdown of the larger social organization, throw men back on the primitive instincts and set free that original human animal that is freed from the social restraints and group critique which makes of us civilized men. He lays stress on hate and fear as the attitudes which here predominate and which are antisocial in their influence. His comments on the expressions of these attitudes are illuminating and will have most salutary influence where read.

There is one conception largely used by Dr. White on which the reviewer cannot forbear a critical comment, and that is sublimation. In the necessarily summary statement in so short a treatise, sublimation appears simply as repression plus the turning of the energies of the repressed instincts into other and socially valuable channels. He thus leaves the reader with the impression, which is otherwise borne out, that man remains at bottom a primitive savage with a group of trained social habits. If these habits break down, the original savage appears. Is not this an inadequate psychologic analysis? Does it not present the training as an entirely external affair? What the reviewer feels this psychology overlooks is the fact that all real training is the result of a conflict of impulses, which leads to reconstruction and reorganization of the individual's own nature. It is only in so far as this inner reorganization is inadequately carried through that man remains at the core a savage. Human nature does not always remain the same. What our Freudian psychology still lacks is an adequate study of the growth of self. But this comment should in no way detract from the impression of the fundamentally sane and highly important message which Dr. White has given out of his professional experience and his experience of the war.

THE ENDOCRINES. By SAMUEL WYLLIS BANDLER, M.D., Professor of Gynecology in the New York Post-Graduate School and Hospital. Price, $7.00. Pp. 486. Philadelphia and London: W. B. Saunders Company, 1921.

In stating his opinion of the book, the reviewer is tempted to borrow some sentences recently used by the Cleveland physiologist, G. W. Stewart (*Endocrinology* **5**:299 [May] 1921), in discussing a certain article: "In reading this paper and many others by 'clinical endocrinologists,' the physiologist can scarcely escape the feeling that here he has broken through into an uncanny fourth dimension of medicine, where the familiar canons and methods of scientific criticism are become foolishness, where fact and hypothesis are habitually confounded, and 'nothing is but what is not.'" Equally apropos is

Stewart's comment on the contrast between the desert in which the physiologists and experimental pathologists have wandered and "the exuberant land of clinical endocrinology, flowing with blandest milk and honey almost suspiciously sweet." Granting that clinical medicine has a perfect right to run ahead of physiology if it can, such an advance is not promoted by almost delirious indulgence in wild hypotheses, of which this book is a product. Those who may be under the psychanalytic spell will here find another world philosophy with its phraseology and also a new basis for interpretation of dreams. We are told that dreams of being frightened mean overstimulation of the suprarenal medulla, dreams of fighting mean overactivity of the suprarenal cortex while dreams of making a speech are due to the anterior pituitary lobe. Childhood impressions and fairy tales are also of importance in Bandler's philosophy on account of their endocrine bearings. He shows that the Freudians are not the only ones who can elaborate a system of symbols. His endocrine symbolism bids fair to outdo theirs both in span and in obscurity.

SYPHILIS UND NERVENSYSTEM. Ein Handbuch in Zwanzig Vorlesungen für Praktische Arzte, Neurologen und Syphilologen. Von DR. MAX NONNE, Oberarzt am Allgemeinen Krankenhaus Hamburg-Eppendorf A. O. Professor für Neurologie an der Hamburgischen Universität und Leiter der Universitats-Nervenklinik. Vierte, Neu Durchgesehene und Vermehrte Auflage. Pp. 1019, with 169 illustrations. Berlin: S. Karger, 1921.

The first edition of this book having appeared twenty years ago, this, the fourth, edition requires little more than a book notice. For many years it has been a standard work and probably is familiar to every reader of the ARCHIVES. The author has made a consistent effort to bring the various phases of the subject quite up to date, and in this he has very well succeeded although, as he states in the preface, unable fully to consult recent literature outside of Germany. In places this lack is so noticeable as to be a real defect. Among the newer questions considered he mentions the curability of general paresis, syphilis of the third generation, family syphilis, the influence of the war on syphilis and the nervous system, and investigations relating to the spinal fluid.

Although this edition is more than twice as large as the first, the division into "lectures" has been retained but the number has been increased from seventeen to twenty. Obviously, the aim of the author has been methodically to cover the entire subject, and this he has done. No aspect of the subject is omitted. Indeed, occasionally there is pedantic fulness at the expense of definiteness. But here and there something is omitted. In a rather full discussion of prophylaxis, including consideration of castration of defectives and criminals, no mention is made of prophylactic applications following illicit coitus.

To his task the author has brought an unusual experience, great industry in assembling the findings and opinions of others and an open mind. An extensive bibliography, an index of authors and an excellent general index close the volume, which should have a place in every neurologic and psychiatric library.